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Transactions

American Medical Association. Section on
Nervous and Mental Diseases. American Medical Association

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TRANSACTIONS OF THE

SECTION ON

Nervous and Mental Diseases

of the
American Medical Association
at the Sixty-Fourth Annual
Session, held at Minneapolis,
Minn., June 17 to 20, 1913

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LIST OF OFFICERS

This Section has been in existence under its present name since 1900. The branches of the practice of medicine which comprise its province were classified with some other subjects in the early days, as is shown by the following section names. The list of officers has been compiled from the published records, which are not complete—for example, the city is not given in some cases. It will be appreciated if any additional information is brought to the attention of the Secretary of the American Medical Association.

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PROCEEDINGS OF THE SECTION

Tuesday, June 17—Afternoon

The meeting was called to order in Folwell Hall, Room 311, at 2:25 p. m., by the vice-chairman, Dr. W. W. Graves, St. Louis.

Dr. Howell T. Pershing's Chairman's address on "Neurasthenia an Increased Susceptibility to Emotion," was read by Dr. C. Eugene Riggs, St. Paul. Discussed by Drs. A. A. Brill, New York; Joseph M. Aikin, Omaha; L. Harrison Mettler, Chicago; D'Orsay Hecht, Chicago, and Howell T. Pershing, Denver.

Dr. T. D. Crothers, Hartford, Conn., read a paper on "Premonitory Auras in Alcoholic Neuroses." Discussed by Drs. A. A. Brill, New York; Charles R. Ball, St. Paul, and T. D. Crothers, Hartford, Conn.

Dr. D'Orsay Hecht, Chicago, read a paper on "Multiglandular Syndromes and the Nervous System, with Lantern-Slide Demonstration." Discussed by Drs. A. L. Skoog, Kansas City, Mo., and D'Orsay Hecht, Chicago.

Dr. Charles R. Ball, St. Paul, read a paper on "Diagnosis, Prognosis and Treatment of General Paresis." Discussed by Drs. C. Eugene Riggs, St. Paul; S. S. Glasscock, Kansas City, Kan.; Lawrence B. Pillsbury, Lincoln, Neb.; Albert E. Sterne, Indianapolis; A. L. Skoog, Kansas City, Mo.; George A. Moleen, Denver; E. E. Southard, Cambridge, Mass.; W. W. Graves, St. Louis, and Charles R. Ball, St. Paul.

Dr. H. D. Valin, Mankato, Minn., read a paper on "Pathologic Findings in Insanity; Illustrated with Lantern-Slides Showing Gross and Microscopic Lesions." Discussed by Drs. E. E. Southard, Cambridge, Mass., and H. D. Valin, Mankato, Minn.

Wednesday, June 18—Morning

Dr. S. P. Kramer, Cincinnati, read a paper on "Recent Advances Made in Study of the Circulation of Brain and Spinal Cord." Discussed by Dr. A. L. Skoog, Kansas City, Mo.

Dr. Julius Grinker, Chicago, read a paper on "A Tumor of the Hypophysis in Acromegaly; a Clinical and Post-Mortem Report, with Photomicrographs." No discussion.

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Dr. L. Harrison Mettler, Chicago, read a paper on "Symptomatology of Multiple Sclerosis." Discussed by Drs. C. D. Camp, Ann Arbor, Mich.; Julius Grinker, Chicago; D. S. Booth, St. Louis; Charles W. Hitchcock, Detroit, and L. Harrison Mettler, Chicago.

Dr. C. D. Camp, Ann Arbor, Mich., read a paper entitled "Epilepsy and Paresis in Railway Engineers and Firemen." Discussed by Drs. C. Eugene Riggs, St. Paul; C. B. Woodson, St. Joseph, Mo.; D. S. Booth, St. Louis; L. Harrison Mettler, Chicago; W. Brown Ewing, Salt Lake; Sanger Brown, Chicago; Leo M. Crafts, Minneapolis; Lawrence B. Pillsbury, Lincoln, Neb.; Charles Gorst, Mendota, Wis.; Charles W. Hitchcock, Detroit; George A. Moleen, Denver, and C. D. Camp, who, in closing, offered the following resolution which was adopted:

That the Secretary be asked to communicate to the Council on Health and Public Instruction of the American Medical Association the fact that the Section believes it is highly dangerous that epileptics and paretics be employed in the operation of railroad trains, and that measures should be taken to prevent the employment of such persons.

The Chairman announced the appointment of the following members to act as committee on nominations: Drs. L. Harrison Mettler, Chicago; Charles R. Ball, St. Paul, and Ross Moore, Los Angeles.

Wednesday, June 18—Afternoon

Dr. E. M. Hammes, St. Paul, read a paper on "Two Cases of Circulatory Disturbance of the Brain," by Drs. C. Eugene Riggs and E. M. Hammes, St. Paul. Discussed by Drs. Sanger Brown, Chicago; Charles R. Ball, St. Paul; H. E. Conley, Cannon Falls, Minn.; Ross Moore, Los Angeles; C. Eugene Riggs, St. Paul, and E. M. Hammes, St. Paul.

Dr. A. A. Brill, New York, read a paper on "The Conception of Homosexuality: Its Theories, Psychologic Mechanisms and Treatment." Discussed by Drs. D'Orsay Hecht, Chicago; Howell T. Pershing, Denver; Ross Moore, Los Angeles; Albert E. Sterne, Indianapolis, and A. A. Brill, New York.

Dr. W. W. Graves, St. Louis, read a paper on "Can Rabbits be Infected with Syphilis Directly from the Blood of Paretics? Observations on the Recognition of the Virus in the Later Periods of the Disease." Discussed by Drs. Charles R. Ball, St. Paul; Sanger Brown, Chicago, and W. W. Graves, St. Louis.

Dr. Albert E. Sterne, Indianapolis, read a paper on "Exudative Encephalitis." No discussion.

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THURSDAY, JUNE 19—MORNING

The following officers were elected: Chairman, Dr. W. W. Graves, St. Louis; Vice-Chairman, Dr. C. D. Camp, Ann Arbor, Mich.; Secretary (for two years), Dr. George A. Moleen, Denver; Delegate, Dr. C. Eugene Riggs, St. Paul; Alternate Delegate, Dr. E. E. Southard, Cambridge, Mass.

The Executive Committee then consisted of Drs. W. A. Jones, Minneapolis; Edward D. Fisher, New York, and Howell T. Pershing, Denver.

To fill vacancies on the Executive Committee caused by the absence of Drs. Jones and Fisher, the Chairman appointed Drs. C. Eugene Riggs, St. Paul., and E. E. Southard, Cambridge, Mass., to act for the session.

Dr. Ross Moore, Los Angeles, read a paper on "Further Notes on the Preneurasthenic and Preinsane Conditions." Discussed by Drs. C. Eugene Riggs, St. Paul; George H. Moody. San Antonio, Tex.; Philip Zenner, Cincinnati; G. Wilse Robinson, Kansas City, Mo.; L. Harrison Mettler, Chicago; W. W. Graves, St. Louis; Howell T. Pershing, Denver; C. R. Woodson, St. Joseph, Mo., and Ross Moore, Los Angeles.

Dr. Charles H. Frazier, Philadelphia, read a paper on "A Division of the Auditory Nerve for Persistent Tinnitus and Vertigo." No discussion.

Dr. G. Wilse Robinson, Kansas City, Mo., read a paper on "Occlusion of the Posterior Inferior Cerebellar Artery. Report of a Case." No discussion.

Dr. B. W. Sippey, Chicago, read a paper on "Demonstration of Lange's Goldsol Test of the Cerebrospinal Fluid." Discussed by Drs. C. Eugene Riggs, St. Paul; Ross Moore, Los Angeles; Peter Bassoe, Chicago; C. B. Woodson, St. Joseph, Mo.; A. L. Skoog, Kansas City, Mo.; Howell T. Pershing, Denver, and B. W. Sippey, Chicago.

Dr. C. Eugene Riggs, St. Paul, read a paper on "Nervous Phenomena in Pernicious Anemia." Discussed by Drs. George A. Moleen, Denver, and Howell T. Pershing, Denver.

Dr. Ross Moore, Los Angeles, moved that a committee be appointed by the Chairman of the Section on Nervous and Mental Diseases to report in one year as to methods by which the Section may be helpful in the question of the hygiene of childhood with reference to nervous and mental diseases. Motion seconded by Dr. T. D. Crothers, Hartford, Conn. Carried. The Chairman appointed Drs. Ross Moore, Los Angeles, and Philip Zenner, Cincinnati.

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NEURASTHENIA AN INCREASED SUSCEPTIBILITY TO EMOTION

HOWELL T. PERSHING, M.D., LL.D.
DENVER

The neurasthenic is always with us, appealing to our sympathy, knowledge and skill to restore him to comfort and usefulness. His sufferings, however he may exaggerate and distort them, are only too real; his disability is often most serious. We are eager to help him, but his symptoms do not yield to the treatment which would relieve similar symptoms due to other causes, while the indications for general treatment are often vague and uncertain. If we knew the fundamental nature of neurasthenia and could direct all our treatment toward its correction, surely we could do better.

For more than twenty years a large proportion of my daily work has been with neurasthenic patients. The aggregate number of cases has been large, certainly more than a thousand, and each case has been carefully examined and studied, and the results have been recorded, in an effort to ascertain the causes and true nature of the disorder. I have not used the method of psychanalysis but I have made a special effort to encourage and assist each patient in telling his hidden causes of worry and fear. I wish to state certain conclusions from my experience which seem to me to be of great practical importance in making treatment more definite and more successful.

As the term "neurasthenia" has been used to denote nervous conditions accompanying very different diseases, it is necessary to define the class of cases to be dis-

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cussed. Nervousness that is incidental to organic disease of the nervous system, or to an infection such as influenza, typhoid fever or tuberculosis, or to painful local disease, such as gastric ulcer or cystitis, should not be described as neurasthenia; there may be a neurasthenic condition but its importance is secondary to that of the major disease. If after the original disease is cured, however, a neurasthenic condition persists, it is to be regarded as a true neurasthenia. On the other hand, various effects of true neurasthenia must not be mistaken for the original cause. Disorders of digestion, of circulation, of vision, of micturition and of the sexual functions are often mistaken by both patient and physician, for the original cause, when they are really only a part of the consequences of neurasthenia. A delimitation is also necessary on the side of the purely functional nervous diseases. The patient with incipient melancholia, paranoia or dementia praecox is often superficially neurasthenic, but the underlying condition is different in kind. Hysteric patients are generally neurasthenic, but their emotional crises are of a different type and they suffer also from suggestibility and restriction of the field of consciousness, causing paralyses, spasms, anesthesias or other stigmata not found in neurasthenia.

Neurasthenia, then, is a functional nervous disorder, not dependent on organic disease of any kind, not amounting to insanity, not hysteria, manifested by irritability, early onset of mental fatigue and a great variety of unpleasant and to the patient alarming sensations, chiefly in the head but also in various other parts of the body.

When we study these cases as to etiology, the idea of fatigue, especially mental fatigue, is forced on us. The patient's own story is usually clear on this point. Overwork in college, perhaps two years in one, or a full schedule of studies along with the struggle for self-support, is a common explanation. A merchant or

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professional man has worked long hours for years without a vacation; a mother has nursed an invalid child while carrying the burden of household cares, and so on. In a smaller proportion of cases without history of previous overwork, an illness, injury or mental shock seems to have caused the neurasthenic condition; but even here the condition, once established, is described as chronic fatigue; the patient has no relish for his work, cannot stand exertion, is always tired. Indeed, the existence and causal relation of fatigue generally appear so obvious as almost to justify the banal prescription, "You must drop everything and get away" (in Colorado it is "You must drop everything and get to a lower altitude"), a prescription by no means easy to take, although easily given, happily freeing the prescriber from troublesome care or the admission that there is anything beyond his scope.

A critical examination of the facts, however, will soon show that work and its consequent fatigue cannot be the fundamental factors in causation. When circumstances are favorable, the neurasthenic can often do a large amount of physical or purely intellectual work and actually be the better for it. Dr. Hall of Marblehead, Mass., successfully carries on a work cure for neurasthenics. Moreover, patients are commonly worse on Sundays or other holidays, and are often worse in the morning than in the evening. Of those who completely give up work only a small number seem to profit. Some are temporarily improved but relapse on returning to the usual condition; many grow worse and worse the less work they do. Even rigorous rest in seclusion often fails and is sure to fail unless the doctor and nurse do far more than relieve the patient of exertion by ministering to his physical wants. The average neurasthenic does not get rested whether he works or is idle. Why not? Because his feelings of exaustion were originally caused and are still maintained, not by work, but by emotion.

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The essential fact about neurasthenics is that they suffer a far greater number of emotional disturbances of the harmful kind than average persons in similar situations. They are incessantly being annoyed, grieved and frightened. It is not that their emotions are extraordinarily intense; their greatest anger is mild compared with the savage rage of an alcoholic; their depression is not to be compared with the despair of melancholia; they are incapable of the passionate contortions of major hysteria; but innumerable, constantly recurring occasions, which would be of trifling moment to a normal individual, cause reactions of anger, fear or grief such as would be appropiate only to the few occasions which are really significant. The condition is like anaphylaxis. That part of the cortex in which emotional processes start has become sensitized so that perceptions or ideas, such as might occur in the normal brain with little or even no appreciable effect, excite a storm of outgoing currents to disturb and disorder all the functions of the body. This oversensitive condition of the emotional apparatus may also be described in the technical language of physiology as a lowering of the emotional threshold.

JAMES-LANGE THEORY OF EMOTIONS

The significance of this aspect of neurasthenia becomes clear when it is viewed in the light of the James-Lange theory of the emotions. Observation and reflection extending over many years have convinced me that, for the practical neurologist, it is a true theory, although the pure psychologist may be doubtful about some of its implications. According to this theory every emotion consists of three processes whose order is invariable.

1. The occurrence of the exciting perception or idea.

2. An action of the brain, excited by this perception or idea, which sends efferent nerve-currents to the organs throughout the body, causing a change in the action of the organs.

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3. The return of sensory nerve-currents from the disturbed organs to the sensory areas of the cortex, causing a change in the sensations and a consequent perception of the bodily disturbance.

Take as a simple example the case of a person startled by sudden noise. The first stage is simply the perception of the sound; the second is the jerking of voluntary muscles, the involuntary cry, the sudden catch in the breath, and the jump of the heart; the third is the disagreeable sensations caused by these sudden changes in the body. In such cases the bodily effects and consequent unpleasant sensations are generally greatly in excess of what one would expect from the loudness of the sound. They may sometimes be useful in enabling one to escape from a sudden danger, but are generally quite useless and their exhausting effect is far greater, the duration of activity being considered, than that of any kind of work whatever. So it is with the various phases of anger, grief and fear in the neurasthenic. The exciting idea is generally not such as one would expect to have a marked effect, but it starts an emotional process whose second stage involves, directly or indirectly, every muscle-fiber and gland in the body, changing motion, respiration, circulation and secretion, while the third stage includes bad feelings which further alarm and depress. Biologically this disproportion of effect to apparent cause is explainable by the fact that the emotions are largely identical with the preservative and protective instincts, for which there is a predeveloped inherited mechanism, which normally in case of danger causes prompt and vigorous action toward escape, defense or attack, without waiting for reason to give its sanction. In modern life this mechanism is seldom useful, while its needless action, if frequently repeated, is always harmful. It is this primitive, inherited, powerful reflex mechanism which has become oversensitive and excessively active in neurasthenia.

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Apply these considerations to a typical case of neurasthenia and it is readily explained. Work may have something to do with it, for all work involves fatigue, and it is not to be denied that normal fatigue due to normal work tends to lower the threshold of the undesirable emotions and to inhibit the desirable ones. The effect of work alone is normally slight, however, and is quickly recovered from; a night's, or at most a few days' rest would insure recovery if the work were the only unfavorable factor; but along with the work are occasions for vexation, anxiety and alarm. The neurasthenic through heredity, disease or traumatism has a brain which reacts excessively to these occasions; to the moderate fatigue of useful labor is added the great fatigue of useless emotion; the neurons whose efferent impulses disturb the body become more sensitive, lowering the threshold still further; thus the daily number of emotional disturbances increases. The sensory centers of the cortex in which the emotion ends become more and more fatigued, so the resulting sensations become more and more unpleasant. This change in sensations, especially in those of the head, causes a feeling of changed personality which is strongly suggestive of mental disease, and thus the earlier fears of heart-disease, apoplexy or what not become relatively unimportant before the terrible fear of insanity.

Psychasthenia is also a lowering of the emotional threshold. Its difference from ordinary neurasthenia is in the nature of the exciting cause of the emotional disturbance. In ordinary neurasthenia the exciting causes are the exceedingly frequent occasions of grief, fear and anger, which are perfectly real but so slight that only the abnormally sensitive brain is harmfully affected by them. In psychasthenia the exciting cause of disturbance is merely the mental representation of a cause for emotion which for the patient does not exist

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at all, but which would be amply sufficient to affect a normal brain if it did exist.

Thus a psychasthenic girl passing a house sees some glasses of jelly exposed on a window-sill. The thought comes, "Some one might put poison into that jelly; what if I should do it?" The mere thought provokes such a feeling of horror that she goes home crying. A few weeks later, she takes part in a cooking-school exercise and is vividly reminded of the ideas concerning the jelly, but keeps control until one of the children who has eaten what she helped to prepare begins to complain of illness. Then the mere idea, which she knows to be utterly false, of having put poison into the food provokes a violent outburst of grief. All normal persons have some emotional reaction to mental representations which they know to be purely imaginary, as in reading fiction or seeing a play. Hamlet comments beautifully on the tears of the player who has been reciting the anguish of Hecuba:

> When she saw Pyrrhus make malicious sport,
> In mincing with his sword her husband's limbs, . . .:
>
> Is it not monstrous, that this player here,
> But in a fiction, in a dream of passion,
> Could force his soul so to his own conceit
> That from her working, all his visage wann'd;
> Tears in his eyes, distraction in's aspect,
> A broken voice, and his whole function suiting
> With forms to his conceit?
> And all for nothing! For Hecuba?
> What's Hecuba to him, or he to Hecuba,
> That he should weep for her?

With normal persons, however, the knowledge that the conceit is imaginary always limits the intensity and duration of the emotional reaction. The theater-goer, the novel-reader, the day-dreamer may really tremble, shed real tears, or contract his muscles in righteous anger, but he restrains himself and quickly recovers emotional equilibrium. Not so the psychasthenic. His emotional centers are so oversensitive that

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a purely accidental image of himself as suffering a violent death, committing a dreadful crime or being insane arouses an intense horror. The intensity of the experience fixes it in memory; it becomes associated with almost everything, and the harmful emotion becomes habitual. The important fact in such a case is not the nature of the idea, or how it came into consciousness, or whether or not it has been repressed into the cellar of subconsciousness; these may have some importance; but the great fact is the physical condition of the cortex which permits such excessive and uncontrolled reactions.

TREATMENT

Whether the patient is simply neurasthenic or also psychasthenic, each excessive emotional reaction leaves a condition of instability which is a predisposing cause of the next disturbance; thus the disease perpetuates itself and he is caught in a vicious circle of cause and effect. How can he get out of it? Only by such measures as will temporarily lesson the number and the intensity of the emotional reactions and permanently change the condition of the cortex so as to raise the emotional threshold. The success of any of the innumerable remedies proposed is in proportion to the fulfilment of this indication, and this explains why apparently the same method is a brilliant success in the hands of one man and a total failure with others.

The founder of the rest-cure has always had a most extraordinary genius for reforming the emotional life of his patients. Those who employ what they conceive to be his method without some spark of this genius fail utterly and even make their patients worse. So it is with diet. Enough good food, preferably at short intervals, is essential; but it would be better for most neurasthenics confidently and cheerfully to commit all the dietetic sins possible at an average table than to eat in doubt and fear only the ideally perfect combination of foods. The same principle applies to work, exercise,

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travel, amusements and reading. They will be beneficial if they conduce to confidence and serenity, but harmful if they cause anxiety, fear or irritation.

The task of reforming the patient's emotional habits and restoring a normal balance and control in his cortex is both difficult and complicated. Every bodily or mental change in the patient has some direct or indirect influence which is favorable or unfavorable, but we must select such a combination of the possible favorable influences as will lead to success and at the same time prevent the unfavorable ones as best we may.

I find it convenient to analyze the problem of treatment as follows: 1. What can the physician do to influence favorably the patient's ideas, and thus control his emotions? This includes everything ordinarily denoted by explanation, suggestion and persuasion. 2. What can the patient be taught to do for himself in happily guiding his own mental processes? 3. What can the patient do in training his body to avoid injurious emotional reactions and favor the desirable ones? 4. What can be done by medicines and other agencies to improve the physical condition of the emotional centers.

1. *Influence of the Physician.*—Much may be done to displace false and depressing ideas by those that are encouraging and tranquilizing. This part of the treatment begins when the patient enters the waiting-room. No pictures of the dissecting-room or sick-bed, no specimens or surgical instruments should be there; all the furniture and decorations should be suggestive of comfort and peace. It seems trite to speak of the physician's attitude toward the nervous invalid, of the need of an interested, sympathetic, patient, confident manner. Yet only a few seem to realize how the patient is influenced by it. Something like "the insolence of office" is easily acquired in hospital and dispensary work; neurasthenics are trying patients and the physician easily slips into an attitude of indifference or exasperation. It is difficult

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to exaggerate the advantage of putting the patient at his ease in the very beginning and making it easy for him to tell his troubles and their real or imaginary causes.

In the anamnesis all the causes of fear and worry should be brought to light, not to magnify but to minimize their importance. I do not believe that these are concealed from the patient himself, but that through fear or diffidence he may conceal them from the doctor, and this is always an obstacle to success. Sexual troubles, whether real or imaginary, should be faced in order to remove them or to show how unimportant they are. The fear of insanity should always be uncovered and then lightly explained as one of the ordinary and almost inevitable symptoms of neurasthenia which does not indicate any real danger and is sure to disappear.

Then comes the physical examination, so careful and complete that the patient feels sure nothing could be overlooked, with reassuring words here and there as one organ after another is found to be sound. In summing up, much satisfaction should be expressed at finding the nervous machinery intact and ready to work well. It may be compared to a good automobile which needs only an adjustment of the spark or carburetor to make it work perfectly, or to an erratic watch which will keep accurate time when the balance-wheel is demagnetized. There must be no hedging in giving a favorable prognosis, but it should be understood that time is necessary to a cure and that after considerable improvement an apparent relapse is sure to occur, which, however, will be only slight and transient if the patient follows directions. The special examinations and reassurances must be patiently repeated as occasions arise. When the patient comes back discouraged and argues that he is no better and that his case is more serious than at first supposed, he really wants to be shown that he is wrong and hopes that the doctor will encourage him. In such cases a gain of a pound or two in weight or any other

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favorable indication may be quite sufficient to put him again on the right track. To agree with him or to be angry at his foolishness is to lose a golden opportunity.

2. The Patient's Control over Emotions.—The patient must be taught to avoid the occasions for unfavorable emotions. He is to shun all controversies, conversations about health or disease and writings on medicine or hygiene whether good or bad. He must absolutely cease studying his own case. His greatest difficulty will be in attempting to control his own trains of thought, especially when morbid sensations suggest them. To this end all harmless measures of diversion are to be cultivated, including both work and play. If reading has been abandoned it is to be resumed, for short periods at first, in spite of apparent bad effects which are due to fear. The patient must treasure up agreeable things to think about and be ready to take refuge in such thoughts whenever his bad feelings are most troublesome and other diversions are impracticable. In directing this part of the treatment a physician can utilize all that he knows of life in both sexes, at different ages and in the various classes.

3. Physical Control by the Patient.—With all possible care, nevertheless, there will be a residue of unavoidable exciting causes of harmful emotions. What can be done to minimize their effects? Each emotion disturbs internal organs which are beyond direct voluntary control, but it also has characteristic effects on posture, movement, expression of face, respiration and voice, all of which are more or less under voluntary control. By sufficient exercise of will on the part of the patient, therefore, an emotion, although already started, can be changed. Thus with a clear idea of what he wants to do the neurasthenic can substitute relaxation for tension, full regular breathing for irregular gasps, the expression of deliberate ease for that of anxious hurry, and an agreeable response for a sharp retort. In doing this he makes a great change in the sensations which consti-

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tute the third stage of the emotion; he to a considerable extent substitutes the sensations of courage for those of fear, of good humor for those of anger, and so on. But this is not all, the internal organs not under direct control of the will are favorably affected indirectly; thus, regular breathing tends to slow and steady the palpitating heart. It is by this process that we learn to overcome the habit of crying in childhood, that we are cured of stage-fright and that we learn to face trying situations of any kind. Note that it is a process, not merely of repression, but of intelligent substitution which can become habitual and be made to prevent even the violent paroxysms of major hysteria. The patient should be drilled especially in correct breathing, so that when he is threatened with a disturbance he can apply the remedy. Then let him start each day with the pious intention to show no fear or vexation in word or expression, still less in action, to make his breathing regular whenever it is disturbed and to keep his voice agreeably modulated and low pitched.

4. *The Use of Drugs.*—Application of the foregoing principles will greatly help all neurasthenics and for some it alone will be sufficient. We have patients, however, who, in spite of all explanations and encouragement, of all possible shelter from occasions for emotion and of honest attempts to practice the expression of repose, serenity and courage, are still habitually fatigued by emotional disturbances. The emotional centers are chronically irritable. They will gradually improve under the plan already indicated and whatever improves general health will help, but the process is slow. In the meantime is there nothing to soothe these irritable centers and so enable the patient to prevent far more of the emotional reactions then he otherwise could? If you can assure him that there is, he will follow your directions with confidence and be more likely to succeed in the mental and bodily training.

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The striking effect of certain chemical agents on emotions leads us to believe that in principle, at least, the emotions can be controlled chemically. Witness the depressing effect of the toxins of influenza and typhoid, the soothing effect of opium, and tobacco, the cheer of coffee and the positive exhilaration of alcohol and various ethers. Alcohol would be a priceless boon if its good effects could be secured and sustained without any disadvantage. It is a pity that its bad effects are so great and so certain. Tobacco and coffee may do some good without serious disadvantage, and I think it a mistake to forbid them to a neurasthenic already accustomed to them; but their good effect is not very great. Bromids, so valuable in epilepsy and so much used in various forms of nervousness, seem to me to make neurasthenia worse. The known relation of the internal secretions to emotion, of the thyroid substance to fear, of the testicular and ovarian substances to amorous feeling, and of the pituitary and possibly the pineal bodies to the sexual glands, warrants the hope that remedies of this kind may be of great use; but so far it is only occasionally that they are of service.

The one remedy whose effect is specific, highly beneficial and capable of being sustained without necessarily involving any serious disadvantage is opium. Far be it from me to encourage its needless or reckless use, but neurasthenia is a source of much unhappiness, often exhausting our resources to the uttermost. If it could be cured by a major surgical operation, the attendant danger of mutilation or death would not prevent the operation from being welcomed by many physicians and many patients. It is not wise to reject the most valuable drug now available, provided skill and care can make it safe. Surgeons do not limit themselves to blunt instruments because sharp ones are capable of doing harm, nor do they reject an operation because an unskilful or careless operator would make it a disaster. The following precautions must be taken: 1. Administration is to

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be only at regular times, never hypodermically but preferably in a capsule, allowing the patient no discretion to increase the frequency of the dose. 2. Other remedies should be combined with it, particularly strychnin and laxatives which will make trouble if the patient exceeds directions. 3. Every prescription should be for a limited number of doses and should carry a prohibition, printed or written in ink, against refilling without special permission. 4. The doses should be small at first, say 0.006 gm. (1/10 grain) of the extract of opium four times a day, and should be cautiously increased, always kept the smallest that will permit a fair degree of progress and later gradually diminished.

Used with these precautions, opium lessens irritability, fear and despondency in neurasthenia, just as it lessens cough and pain in other diseases, and by permitting emotional rest it gives the exhausted nerve-centers a chance to recuperate and greatly aids the patient in forming the correct emotional habits which he is being taught. I am sure that I have succeeded with it in many cases in which without it the difficulties would have been too great; I have never known a drug habit to follow its use in this way, nor have I had any difficulty in discontinuing it when it seemed best to do so. The dose need not be large; most patients do best when the maximum dose reached does not exceed 0.02 gm. or ⅓ grain of the extract. Only in a few desperate cases have I reached the larger doses such as would be suitable in melancholia, sometimes being rewarded by complete success, sometimes failing.

I know that many will think that in this discussion of neurasthenia I have treated slightingly or altogether omitted the most important factors, such as digestive disorders, ocular defects, defective elimination, pelvic diseases, sexual errors and so on. Any one of these factors may be of great importance on its own account or as a complication, but so far as the neurasthenia is concerned I regard them as secondary or incidental, one

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might almost say accidental. They have been disregarded in order to show more clearly and in their true proportions the essentials of neurasthenia.

Stedman Building.

ABSTRACT OF DISCUSSION

DR. A. A. BRILL, New York: Dr. Pershing is trying to reduce the conception of neurasthenia to some definite symptom-complex. He seemed, however, in the course of his discussion, to take in conditions which go beyond the scope of the definition with which he started. For instance, despondency and depression do not necessarily belong to neurasthenia, unless we take neurasthenia to include almost anything. I was especially interested in the statement that the symptom of fatigue in neurasthenics is more pronounced in the morning. In my own experience I have found that neurasthenics usually feel much better in the morning and become more and more fatigued as the day advances. On the other hand, patients with depression of the manic depressive type usually feel much fatigued in the morning and, as the day goes on, the fatigue wears off. I should be interested to hear some discussion on this point. If time permits, I should like to hear discussed the James-Lange theory as applied by Dr. Pershing to neurasthenia, which I must say I cannot accept. I was pleased to hear that Dr. Pershing included the mental element in the treatment, for to me psychic treatment is most important in neurasthenia. I would caution against the use of morphin in neurasthenics. I never use it in any of my cases.

DR. JOSEPH M. AIKIN, Omaha: I want to endorse Dr. Pershing's statement with reference to the symptoms of early morning fatigue. Until recent years I regarded that as more particularly a part of the clinical picture of melancholia, in which the depression is so much greater in the early morning than in the later hours of the day. I have, however, in the last three or four years been able to verify the particular symptom of which Dr. Pershing speaks. I am especially pleased that Dr. Pershing does not endorse the use of the bromids in neurasthenia. I have tried them out pretty thoroughly, and do not approve of their use in neurasthenics. Dr. Pershing also voices my experience in rejecting any form of opium whatsoever in these cases. I do not use a hypodermic on a neurasthenic, for any opiate at least, and I avoid, if possible, its employment for any drug whatsoever. There is something in the needle habit that we ought to avoid, even though there be nothing but distilled water in the syringe. What Dr. Pershing presented with reference to

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laxatives is in line with my experience in the treatment of neurasthenic cases. The less we prescribe laxative medicines or any drugs in the treatment of neurasthenics, the better our results. Intelligent direction as to quality, quantity, time and manner of taking food will give the patient more relief than drugs. If we can get these patients to eat a good variety of food and forget what they are eating, the necessity for laxatives will soon disappear.

DR. L. HARRISON METTLER, Chicago: If we are going to limit the term "neurasthenia" to mere psychic reaction of the emotional type, Dr. Pershing's presentation of the management of neurasthenia is most careful and practical. I am not ready, however, to accept the idea that all of the cases that I have minutely examined and conscientiously denominated neurasthenia are altogether psychic in character, the expression of merely an exalted affective psychosis. Remembering the elaborate and painstaking experiments of Hodge, Vas, Mann, and others in the laboratory, I have remarked a close similarity between the symptomatic revelations in the animals experimented on and the patients in whom I have made the diagnosis of neurasthenia. It is impossible for me to believe that emotional feeling is the basis of these animalistic revelations, as I am asked to believe that they are in the human being. Again, I have always noticed that in the incipiency of almost every infectious disease, long before the disease can be positively and definitely diagnosed, the clinical picture bears a remarkable similarity to that of the fatigue neurosis which we have been wont to call neurasthenia. Here I assume that the trouble is assuredly greater than a mere hyperemotionalism.

To sustain his position, Dr. Pershing adopts the James-Lange theory of emotion, wherein the feeling is said to follow, and not to precede, the cardiac, vasomotor and other disturbances. This theory I have never been able to accept. It is incomprehensible to me that a horrible sight, for instance, should cause the vasomotor apparatus to be disturbed and that this disturbance then causes the feeling of horror. The basis of the horror is not the mere thing looked at, but the ideas springing out of it. Without these ideas the sight would be wholly neutral and awaken no emotion. Once awakened, the emotion in its force and suddenness stirs the cerebral centers, and these reflect their disturbance on all of the organs of the body.

The James-Lange theory might apply in a way to the emotional states of neurasthenia as of any other disease. Abnormal conditions of the body always affect more or less the patient's mental state, but this is not what I understand that these authors intended to convey in their explanation of the emotions. At all events, if I am wrong about

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this, this application of the theory upholds my contention that neurasthenia is something more than a mere psychosis. I find the psychosis to be the result of some organic disturbance within the body in most of my neurasthenic patients. Psychotherapy alone, useful as it is in hysteria and other pure psychoneuroses, does not help me in the management of neurasthenics. I find that I have to resort to hygienic, physical and medicinal measures of all sorts to restore the patient's bodily health; then the mental state improves *pari passu*.

Dr. D'Orsay Hecht, Chicago: Although I am fully in accord with many of the views expressed by Dr. Pershing in his address, I must confess that I was anticipating something a little different in the attitude toward the general broad conception of neurasthenia as a disease entity. I had hoped to hear an expression of the same regret that I have felt for so long, that we, as neurologists, had too tenaciously clung to the view of neurasthenia as a distinct clinical condition. I, for one, am interested in the passing of neurasthenia as a disease *per se* in the sense in which Beard put it forth. I would even venture to add that, in my opinion, the whole tradition of neurasthenia as a primary condition of irritable weakness should have perished long before ever it was well on its way in medical literature. I have come to believe that in practically all instances of so-called neurasthenia a careful physical examination will reveal underlying pathology, perhaps not obvious pathology, but enough of organic change, however latent or subtle, to render the fatigue phenomenon explicable. If we look at neurasthenic individuals, moreover, as sick primarily in their bodies and not in their minds or their "nerves," we shall have gone far toward a better understanding of an asthenic symptom-complex. Hitherto we have put too much stress on the emotional values in these cases, and have seriously confounded them with the psychoneuroses, with which, I believe, they have little or nothing in common. I sometimes wonder if we are sufficiently careful to determine a small half-hidden focus of tuberculosis, which a tuberculin reaction might reveal. Again, I wonder if polyglandular insufficiencies are sufficiently appreciated to be held accountable for a fatigue syndrome. I wonder whether the rôle of faulty metabolism is well enough understood to be thought of in this same connection. Surely our increasing refinement in the field of serum and vaccine therapy should pave the way not alone for greater accuracy in diagnosis, but also for better success in the treatment of these patients, with their reduced vitalities, chronic, vague pains, nervous irritability and other evidences too familiar to recount. It is the physical diagnosis that I regard as important in the type of cases to which we refer, and the more keenly that we regard arteriosclerosis, latent tuber-

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culosis and low grades of sepsis from one source or another, and what, for lack of a better term, we may call the toxemias of polyglandular origin, the less will be our provocation for making the diagnosis of neurasthenia.

DR. HOWELL T. PERSHING, Denver: I admit that any definition of neurasthenia is only approximate. I must also confess that I have occasionally made the mistake of regarding incipient melancholia, exophthalmic goiter or pulmonary tuberculosis as neurasthenia. As time has gone on, however, and I have done my best to perfect my diagnostic methods, so as to exclude all such conditions, there still remains a large number of cases of what in our present state of knowledge can be called only neurasthenia. Even the finding of some organic lesion does not forthwith exclude neurasthenia. There is a neurasthenic type of patient, with slight organic disease, who is quite different from the average patient with the same disease; the emotional disturbance is so out of proportion to the physical disorder that the neurasthenic condition must be dealt with on its own account.

I admit, of course, that neurasthenia, once established, has a physical basis in the oversensitive cortical centers, and that this may be aggravated by many morbid conditions in various parts of the body; but the essence of my contention is that exciting causes, which we are in the habit of regarding as purely mental, have far-reaching bodily effects, which not only aggravate but also originate and perpetuate the neurasthenic condition. That mere perceptions, mere ideas (the physical basis of which is purely cortical), do produce extensive and profound bodily effects is a matter of commonsense observation. Else, why should a piece of bad news cause sobs, tears, tremor, palpitation, arrest of digestion and loss of weight? Let us by all means study the physical condition of the patient from every point of view and improve it to the utmost. If we narrow our vision to the physical side, however, and do not improve the patient's mental habits we fail. In a case of nervous, that is neurasthenic, dyspepsia, you may wash the stomach, make the diet perfect and give any or all of the aids to digestion, with the result that the patient is still a dyspeptic and may be even worse. Leave the stomach alone, correct the emotional habits and you cure him.

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THE NATURE, DIAGNOSIS, PROGNOSIS AND TREATMENT OF GENERAL PARESIS

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The honor of having first discovered a relationship between paresis and syphilis belongs to Esmarch and Jessen. These observers in 1857 reported three cases of paresis, the cause of which they attributed to syphilis. Thus a controversy of this subject began which has lasted for more than half a century. The consensus of medical opinion has held that in the great majority of cases syphilis is the chief etiologic factor which causes paresis. Of the opponents of this doctrine Näcke was the most extreme. He considered a neuropathic inheritance as a most important cause and assigned to syphilis an equal importance among other etiologic factors, maintaining that it often furnished the last offense for the development of the disease. It was not until the discovery of the Wassermann reaction and its regular observance in both the blood and spinal fluid of paretics that the doctrine of no syphilis, no paresis, could be said to have found universal acceptance.

Until recently parasyphilis has been regarded as an effect of syphilis, the ashes of a fire which long ago has burnt itself out, the result on the nervous system of a toxin which might be compared to the action of the toxin of diphtheria in diphtheritic paralysis, when the exciting factor, in both instances, had passed away, but the harmful influence still acted in a progressive manner.

A number of reasons may be given why we have so persistently refused to accuse the spirochetes of occupy-

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ing the chief rôle in the causation of the symptoms of paresis. The apparent absolute inefficiency of antispecific treatment, the fact that the pathology of paresis was said not to be the pathology of syphilis and the failure to discover the spirochetes in the tissues of the central nervous system are the chief reasons. Since the discovery of the Wassermann reaction, however, and its positive finding regularly in paresis, the suspicion has steadily grown that the *Spirochaeta pallida* is actively and directly concerned in paresis. Plaut calls attention to the fact that a paretic behaves with reference to the serum reaction similarly to a syphilitic in the active stages and not as a tertiary luetic without symptoms. This indicates, according to Plaut, a more active participation of the spirochetes in paresis than was formerly thought. He believes that there is a close relationship between the activity of the specific virus and the Wassermann reaction. Nonne says that occasionally the blood of a paretic may give a negative Wassermann and that rarely also all four actions may be negative in tabes. These results usually occur, however, in exceedingly long-standing and chronic cases after the active process and progression of symptoms have long since terminated. This observation of Nonne's would seem to support Plaut's contention.

The fact that active syphilitic processes in the nature of gummata and specific endarteritis are found both in paretics and in tabetics also argues for a close connection between the spirochetes and paresis. Recently E. Meyer[1] has reported a case of syphilitic meningomyelitis occurring in a paretic. The active syphilitic process in the cord was healed by antispecific treatment, as the post-mortem later demonstrated.

The pathology has been one of the strong arguments against regarding paresis as a late syphilis. On this

1. Meyer, E.: Progressive Paralyse kombiniert mit Meningo-Myelitis marginalis, Arch. f. Psychiat. u. Nervenkrankh., L, Band 50, Heft 1, Seite 245.

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question Spielmeyer[2] says that, although the pathology of paresis is a characteristic one and differs materially from the pathology of cerebral lues with which we are familiar, we cannot say that it is no longer a syphilitic process. He argues that as we are unable to say with certainty what pathologically belongs to syphilis, therefore we surely are not justified in saying that certain processes are non-specific in character. Primary parenchymatous degenerations can just as easily be caused by syphilis as specific cellular infiltrations. Nissl takes a somewhat similar position. Thus according to these observers we are not able to state positively that the pathology of paresis may not also be the pathology of syphilis.

The inefficiency of antispecific treatment has been another important reason why the diseased processes of paresis have been regarded as of a different nature from those of lues. The tendency to exclude syphilis when a disease failed to respond to antispecific therapy has been too general. We have known for some time that certain specific processes are often refractory to treatment. Nonne[3] says that the general medical opinion in regard to the success of antispecific therapy is too optimistic. Spielmeyer suggests as an explanation of the failure of antispecific therapy the impermeability of the meninges. A large number of experiments have been made and from these it has been demonstrated that mercury and iodin, under normal conditions, are unable to penetrate the meninges. The difficulty exists in paresis and tabes. This author says that perhaps the inadequateness of our present methods of treatment in not being able to pass through the meninges may be largely responsible for the failure of this treatment in paresis.

2. Spielmeyer: Die Behandlung der progressiven Paralyse, Arch. f. Psychiat. u. Nervenkrankh., Band 50, Heft 1, Seite 76.
3. Nonne: Syphilis and the Nervous System, American edition.

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Recently Noguchi[4] has examined the brains of a large number of paretics. Out of 200 examined he has been able to find the spirochete in forty-eight, and in the spinal cord of one tabetic out of twelve examined. He found the spirochetes more numerous and more frequent in the brain cortex than in the white substance. They were distributed between the nerve-cells and the neurogliar tissue. In some instances the parasites were found in the nerve-cells. In those cells containing the spirochetes evidences of degeneration in the form of distortion and swelling, as well as the complete dissolution of both the nucleus and the cell-processes, were discovered. Noguchi, in connection with the report of his examination, says that the pathogenesis of paresis was never entirely free from objection. Since, however, the causal factor of syphilis has been found in the pathologic changes, one may well assume that these changes are directly due to the *pallida*. He characterizes the brain changes as a chronic parenchymatous encephalitis produced by the invading spirochetes. He suggests that the fact that treatment thus far has proved so futile may be explained by the circumstance that the parasites lie in the nervous parenchyma away from the blood-vessels and are inaccessible to the medication. Confirmation of Noguchi's discovery by other observers is all that is lacking in order to bring about a complete reconstruction of our former ideas concerning the nature of metasyphilis. This must all deeply affect the whole subject of paresis. It increases very much the importance of diagnosis and particularly of an early diagnosis. Kraepelin[5] says that it is often very difficult to determine the actual beginning of the affection. A number of years may intervene between the observance of such symptoms as transient attacks

4. Noguchi, H.: Studien über den Nachweis der Spirochaete pallida im Zentralnervensystem bei der progressiven Paralyse und bei Tabes dorsalis, München. med. Wchnschr., April 8, 1913, Seite 737.

5. Kraepelin: Psychiatre: Die Dementia paralytica Verlauf; Ausgang, Ed. 8, ii.

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of double vision, dizziness, temporary aphasia and unconsciousness and the real development of the paresis. He reports two cases in which there was an interval of six years between the observance of such symptoms and the beginning of the paresis. I can recall one case which came under my observation with symptoms of dizziness, loss of the pupil light-reaction and occasional attacks of unconsciousness in a patient with a luetic history four or five years before sufficient mental change had occurred to make it necessary for the patient to give up his regular work. When should the diagnosis have been made in this case, at the time of the first attack of unconsciousness or four years later when the paresis was well advanced? It is interesting and, in the light of our recent knowledge, very important to know how far back we may date the real origin of the paresis with a history of such symptoms as those mentioned.

Another interesting and practical question is, if this case had been regarded as paresis when the first symptoms appeared, would active and persistent antispecific treatment, such, for instance, as the chronic intermittent, or the intensive along the lines proposed by Dreyfus and Gennerich, have exercised a restraining and beneficial influence on the later course? It must be acknowledged that the only difference between tabes and paresis exists in the location of the pathologic process. Because of the larger brain-area and, therefore, greater capacity for compensation, the prognosis for the brain-process ought to be better than for the one in the cord. In regard to the influence of antispecific treatment in tabes, Nonne says that for many years he has had a large number of cases of beginning tabes under observation in which the disease had apparently been brought to a standstill by means of regular courses of inunctions. Because of the peculiar anatomic structure of the cord, beginning lesions there would naturally cause much earlier significant symptoms than corresponding processes in the brain. On account of

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the so-called silent areas in the brain, extensive lesions may exist without manifesting any symptoms. Failure to recognize the nature of early symptoms before the diseased processes have become too extensive may be another explanation why antisyphilitic treatment has proved so ineffectual in paresis.

During the past two years it has been my practice to make a Wassermann examination of the blood in every one of my hospital patients. If the Wassermann was found to be positive and the patient exhibited any nervous symptoms, no matter how slight in character, I then made a lumbar puncture and tested the three reactions. With positive findings the interpretation of even slight psychic symptoms becomes easier. With the modern diagnostic methods a much earlier diagnosis of paresis becomes possible, and the importance of this, in the light of recent additions to our knowledge, is greatly increased.

Nonne, in his book, "Syphilis and the Nervous System," says that a genuine paresis was formerly considered incurable, but that this opinion at the present time cannot be unqualifiedly maintained. Schüle reports a case of undoubted paresis with recovery after an attack of double pneumonia. The patient remained well twenty years. Many other similar cases have been reported. The tendency among medical men to give an absolutely bad prognosis in paresis as soon as the diagnosis is made has been too general. It has been estimated that 10 per cent. of all cases undergo remissions of variable duration, and from 0.5 to 1 per cent. complete and long-continued remission. I remember that several years ago, after treating a case of paresis for a number of months, I gave a bad prognosis to the family and insisted that the patient should be sent to a state institution. Under osteopathic treatment in the course of six weeks the patient so far recovered that he was able to return to work. His remission lasted just about a year. Such remissions are not uncommon and they

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may come at any time. In this connection the case reported by Tuezek is worthy of mention. The patient, in 1876, when 38 years old, manifested typical symptoms of paresis, which in 1878 disappeared. In 1883 he developed tabes without any psychic symptoms, which gradually progressed. In 1898, twenty years after the beginning of his disease, he again exhibited symptoms of mental confusion and excitement along with mental enfeeblement, which two years later caused his death. According to Nissl, who made the necropsy, the pathology of the cortex was that of paresis. This case illustrates well the truth of the old German medical *Sprichwort,* "die Syphilis stirbt nicht, sie schläft nur." In making a prognosis in paresis one should always keep in mind the possibility of remissions.

During the last few years considerable effort has been made to find a suitable treatment for paresis. It has been recognized for a long time that fevers, infections and suppurations, such a pneumonia and erysipelas, often exercise a most favorable influence on the affection. There are many observations in which the disease has been brought to a period of quiescence through such intercurrent affections. According to Metchnikoff the important factor in exerting this beneficial influence is the leukocytosis caused by infections of this kind. Wagner's tuberculin treatment, the injections of mixed cultures of dead bacilli and sodium nucleinate, first suggested by Donath[6] of Budapest, were proposed for the purpose of artificially creating this leukocytosis. For two years I have been using injections of sodium nucleinate in cases of paresis under my care. The sodium nucleinate was given in the following manner: Two and one-half gm. of the drug were dissolved in 100 c.c. of normal salt solution made from distilled water and sterilized. The injections were administered weekly. Forty c.c. were given at the first injection and

6. Donath: Salvarsan in der Behandlung der syphilischen und metasyphilitischen Erkrankung des Nervensystems und dessen kombinierte Anwendung, München. med. Wchnschr., Oct. 22, 1912, Seite 2342.

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the quantity was increased each week by 20 c.c. until 100 c.c. had been injected. Then the weekly dose was further increased by adding 0.5 gm. of the sodium nucleinate to the solution until the maximum dose of 5 gm. was reached. All of the injections were made into the muscles of the nates. In the eight cases so treated, the four reactions were taken at the beginning of the treatment in each case and then again at various intervals during its course. A leukocyte count was also made before and after the injections. At different periods during the treatment an intravenous injection of salvarsan was alternated each week with the sodium nucleinate. The influence of the treatment on the four reactions was somewhat irregular and uncertain and did not always correspond with the clinical symptoms of the patient. For instance, in one case after the salvarsan injection the positive Wassermann in the blood became negative while the condition of the patient was growing worse. In general the effect of the treatment was to lessen the intensity of the reactions. The reactions in the spinal fluid were not influenced to so great an extent as was the blood-reaction. In none of the cases did any of the three reactions in the spinal fluid become negative. From one to two hours after the injection the patient usually had a chill, which was sometimes very severe. This was followed by a rise in temperature, the temperature often going as high as 103 and 104 F. Usually the temperature became normal again in from twenty-four to forty-eight hours. The number of leukocytes was always increased after these injections, sometimes being twice the number they were before twenty-four hours after the injection. In two cases rather severe suppuration and abscess formation followed the injections. In one cases there was a rise of temperature for two weeks as a result of an abscess. In this case at present there is a complete remission. All of the eight cases were well advanced. Two of the patients in this series of cases manifested no improvement whatever, but grew steadily worse. Two

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had short remissions, one of a month's duration, the other of four months, complete enough so that they were able to return home and care for themselves, but not able to resume their occupations. Two patients who showed considerable improvement are still at home and have been for more than a year. One of these had exceedingly severe symptoms. He was confined absolutely to bed, frequently had involuntary urination and stools, had to be fed by tube and did not speak for several months. While in the hospital he developed severe decubitus. This patient received three salvarsan and twenty sodium-nucleinate injections. His remission came rather suddenly. One day he simply waked up—like Rip Van Winkle—and began to talk and manifest an interest in things around him. From being an animal that was just alive, he became something of a human being, clean in his habits, and able to make his own toilet and to go down town and to church alone. His mind was free from delusions, but somewhat enfeebled. A recent letter from his wife states that he is not so well and she fears that he will have to be placed in the hospital again. The other patient received twenty-four injections of sodium-nucleinate, after which he returned home in very fair condition. He remained quite well for six months, then suddenly had a severe attack of convulsions, following which he lost his speech entirely. He was returned to the hospital and this time received three salvarsan and eight sodium-nucleinate injections of 5 gm. each, the last injection being given in December, 1912. An inquiry, May 1, regarding his condition, elicited the information that he had not been so well mentally and physically for years as he is at present. Of the eight patients treated, two are dead, two are in one of the state institutions and four are still at home. Of these four, the mental condition of two is that of a rather moderate simple dementia. The mental and physical health of the other two is excellent.

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This treatment, according to the results which I have obtained, cannot be said to be a great success. It seems, however, to have exercised in varying degree a favorable influence in all the cases with the exception of two. Since the discovery of the Wassermann we have been saying no syphilis, no paresis. We are now able, since the finding of the spirochetes in the tissues of the brain and spinal cord, to go a step farther and say, no spirochetes, no paresis.

These recent contributions to the subject of metasyphilis have taught us especially two things; first, the nature and location of the enemy; second, the great importance of an early diagnosis, if we wish to have a reasonable hope of destroying him.

Lowry Building.

ABSTRACT OF DISCUSSION

DR. C. EUGENE RIGGS, St. Paul: The discovery of the *Spirochaeta pallida* in the brain of the paretic has been the signal for the passing of parasyphilis. There is not the slightest question, I believe, but that paresis is nothing more nor less than a phase of syphilis due to the direct action of the spirochete. I do not mean to convey the idea, however, that the pathologic process in syphilis is the same as that in paresis. In syphilis we have endarteritis, gumma and meningitis; in paresis we have degeneration of the neuron tracts of the spinal cord and of the brain. Why in the one instance we have a degenerative process and in the other a syphilogenous process is a question hard to explain. Possibly the personal equation has something to do with it, or a special toxicity of the spirochetes. Mott cites an instance in which one woman infected five men and every one of those men had paresis. As Dr. Ball has said, we are dealing with a syphilitic process, with the direct influence of the *Spirochaeta pallida* on the nervous system. This gives us a reasonable basis for the use of salvarsan in these cases.

In referring to the early stages of paresis, in which we have vertigo and epileptiform and apoplectiform attacks, Dr. Ball asks whether these symptoms indicate paresis or nervous syphilis. This is a question. The indication is certain, however, for the use of the appropriate remedy in a positive, definite way.

Dr. Ball speaks of a remission of twenty years in one case. There are instances reported of even longer remissions. In referring to the question of recovery he was careful in choos-

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ing his words. I think that this is extremely questionable, because if a really degenerative process once takes place, the degenerated tissue cannot be repaired.

Dr. S. S. Glasscock, Kansas City, Kan.: There are undoubtedly some symptoms of beginning paresis that we have overlooked. In the future we shall be able, though these symptoms be ever so insignificant, to make an early diagnosis of paresis, and, by the use of salvarsan, or some other remedy to be developed later on, to treat these cases at a time when we can cure them. I am inclined to think that this knowledge will stimulate among men who have had syphilis the idea that just as soon as they have any nervous manifestations they should see a man competent to make the diagnosis. Possibly, moreover, if they have not the symptoms of the disease, and these examinations are made, we may be able by the use of salvarsan to eliminate from the system the spirochete, by that means ultimately eliminating locomotor ataxia and paresis.

It would be well for us to consider in regard to salvarsan whether or not arsenic, even though it be in a non-toxic form, given in large doses or administered repeatedly in these cases of syphilis, may not ultimately (when a period of years has passed and paresis or locomotor ataxia may be expected to develop) because of its toxic properties have so profound an influence on the nervous system that this will tend to increase the number of cases of paresis and locomotor ataxia instead of decreasing them.

Dr. Lawrence B. Pilsbury, Lincoln, Neb.: We are badly in need of a critical test for paresis, especially in the early stages. At present I happen to have under my care a man about 65 years of age who came to the hospital with a history of some loss of judgment and some disturbance of emotion. He was jealous of his wife, probably unjustly so, and had a coarse tremor in both hands and a shuffling gait, holding his head forward, but with no definite tendency to propulsion or retropulsion. I felt doubtful as to what I was dealing with until I performed lumbar puncture. Both blood and spinal fluid gave a positive Wassermann reaction. There were 130 cells and increased protein in the fluid. He now has a coarse tremor of about four per second. I suppose that it is a case of paresis. The symptoms began only some six or eight months ago.

Another case was that of a man about 35 years of age whose case I reported to a medical society as typical of paranoid dementia with grandiose delusions pretty well systematized and fairly constant. At that time I could discover no motor symptoms and no physical findings that would lead one to think that he had paresis, but I will admit that I did not puncture him as I should have done. Later on I punctured

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and found an increase in cells and protein. He now has a slight inequality of the pupils, and his face has flattened out somewhat, with just a mere suggestion of tremor of the facial muscles. He is neat and clean and industrious on the ward.

Another case was that of a man of about 44 years of age who was formerly in the hospital as an undoubted case of manic-depressive insanity in the depressed phase. Twenty years ago he recovered and went away. He had a manic-depressive family history. One of his sisters is in the hospital now and has been there repeatedly. A year before he came to the hospital the last time an internist diagnosed the case as paresis. I thought he was mistaken and so stated to the patient and his friends. The patient had a well-marked hypomania and there was no amnesia that could be discovered. I punctured him at that time and failed to find any increase of cells, but discovered a slight increase of protein, which I disregarded. Later on I punctured him again and found an increase of both cells and protein. The pupils were definitely unequal and unequally responsive to light. They became so rather suddenly, for I had examined him from time to time.

DR. ALBERT E. STERNE, Indianapolis: Of course, we all recognize metasyphilis as syphilis, and the condition should be treated as syphilis as early and as vigorously as possible. From some remarks that have been made one would infer that the pathology of paresis is confined to the nervous system. It is chiefly in the nervous system as neurologists see it, but there is scarcely an organ in the body that is not involved. In quite a number of cases, moreover, we find that the condition involves the central nervous system preeminently. These changes we meet in syphilis. I believe that Dr. Riggs remarked that in metasyphilis we have no endarteritis, but to this I do not agree; the secondary changes never could be influenced by the specific treatment so far as we know at the present time. Again, we must consider as coming under this category those cases of paresis which run a clinical course quite different from those that we usually see—those cases in which mental symptoms are manifested, cases of progressive dementia, without the usual euphoria, and without delusions, cases, so to speak, that fade away with almost perfect recognition on the part of the patient that he is sick. Ordinarily by the term "metasyphilis" we designate only cases of paresis, but etiologically it is of importance in other organic psychoses. We are now engaged in a careful examination into the rôle of syphilis in the development of dementia praecox, and I am anticipating a report to be made next week at the meeting in Chicago in which we shall show a positive etiology of syphilis in about one-third of the cases of clinical dementia praecox. It seems to me that in these cases, as in syphilis, we should not neglect the mixed treatment. While we may rely to a

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considerable degree on salvarsan, we should never neglect the adjuvant treatment which we have always used. This treatment, moreover, especially in paresis, should be given with a great deal of caution, as in my experience the use of salvarsan in these cases is not without danger. In some cases we are now using serosalvarsan, that is, blood-serum plus salvarsan, by intraspinal injection. The results are as yet too problematic to offer more than encouragement to the continuation of this particular type of treatment.

DR. A. L. SKOOG, Kansas City, Mo.: I would question the statement, "No syphilis, no paresis." Possibly reactions have not always shown up positively. There is a certain small percentage of cases in which tests give negative results. Investigators have, as stated, found spirochetes in less than 25 per cent. of cases of paresis. So this can hardly be used as a strong argument. Clinically, however, I believe that all patients with paresis originally suffer directly or indirectly from syphilis.

DR. GEORGE A. MOLEEN, Denver: I would call attention to a point brought out by Dr. Pilsbury, which seems to have caused some surprise on the part of writers in the foreign journals: the question of isolated syndromes, such as the slow tremor. A case mentioned in the Revue neurologique (1911, xxii, 123) was taken to be one of paralysis agitans. In my own experience, a man presented a tremor, confined to the right hand, which could hardly be mistaken for anything but paralysis agitans. He presented little mental disturbance and later developed some mild persecutory ideas, as a result of which he was placed in a general hospital and observed. The tremor has continued and his mental condition has been limited to the persecutory ideas, but within the past two weeks he had a very typical apoplectiform seizure, and I think that there can be no question as to the diagnosis of paresis. The pupillary retardation could be determined only if compared to the reaction of distance, and that is often the case with those symptoms which one might consider as part of the syndrome. I think the pupil should be studied by comparing the rapidity of its reaction to light with that to convergence. Since seeing the case reported in the foreign literature, however, and hearing Dr. Pilsbury speak of it, I am reminded of my own, and thought best to recall the case here as presenting symptoms which sometimes lead to error in the making of a diagnosis of paralysis agitans, when the patient might have beginning paresis as indicated by such an isolated symptom. In my case there had been a positive Wassermann, and besides, there was a distinct, clear history of luetic infection ten years previously, treated for two years by a competent internist.

DR. E. E. SOUTHARD, Cambridge, Mass.: If I am not mistaken we have a similar case at the hospital at present. The

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patient has the signs in general of paralysis agitans except tremor of the right hand. He had a positive Wassermann reaction, with a total negative finding.

In this connection I might say that at the Psychopathic Hospital statistical data show something that I should like to have explained. We have been making a monthly count of Wassermann reactions up to 31.5 per cent. in a class of cases which are perhaps a little different from the ordinary cases. In the state hospitals of Massachusetts 22 per cent. of positive Wassermanns were found in the total count of the hospitals, including paresis and all the other cases admitted. In a good many cases of dementia praecox we shall, I believe, find that we have had a positive Wassermann reaction. Whether this indicates any etiologic relationship between the *Spirochaeta pallida* and the dementia praecox is dubious. Dermatologists used to say that about 15 per cent. of their infirmary practice was specific. This has to be taken into account when we draw conclusions as to the relation of the Wassermann reaction to paresis. We do not feel that there are any exceptions to the positive Wassermann in syphilis. It has been shown that cerebellar sclerosis is not incapable of making a condition resembling that of general paresis, which may account for the few cases in which it has been absent. In a series of forty-one cases studied, two were cases of cerebellar sclerosis. There might have been positive Wassermann reactions in those cases.

Dr. E. E. Southard, Cambridge, Mass.: The spirochetes certainly do not inhabit the neighborhood of the vessels, and while this organism may not be responsible for all the lesions in paresis, the localization of the lesions in the brain in paresis would seem to favor this idea. One wonders, of course, whether in various other diseases the symptoms may not be due to death of the spirochetes rather than to any factors connected with the living organisms. In one case the effects were traced to the cerebrospinal sheath. Pain occurred as a result of treatment in a patient in whom pains had never before occurred. We wondered, therefore, whether this result might be due to toxic elements, or to the killing of the spirochetes. One must take into account the endotoxic results of the killing of the organisms by salvarsan. Some of the results of the so-called provocative injections of salvarsan may be due to these endotoxic effects, the spirochetes being killed by the provocative dose, as the result of which there are symptoms which may then be succeeded by an improvement.

Dr. W. W. Graves, St. Louis: I have long held the opinion that without syphilis there is no paresis and no tabes. That conclusion is based on the observation of others and also on personal experience, extending over a number of years of observation not only of syphilitics whom I treated many years

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ago, but also of their wives and children. Not only do almost 100 per cent. of Wassermann reactions on the blood in paresis lead to that assumption, but the clinical signs also, which are invariably present when associated with laboratory signs, cause us to come to the same conclusion. During the past three years I have endeavored to recognize, if possible, by microscopic and other means, the virus of syphilis, and I shall be able to present at this meeting some evidence along lines showing that it is possible to infect rabbits with the virus of syphilis direct from the blood of general paretics.

There is one other point to which I should like to refer, one I have observed not infrequently in connection with the Wassermann reaction in general paresis. It is known that a certain number of paretics will give a negative Wassermann. This finding seems to depend somewhat on the time the Wassermann test is made; if immediately following a paretic attack, or during or coincident with the attack, the reaction will not infrequently be negative, while if made during the remission the reaction is almost invariably positive. I interpret the phenomenon in this way: The system is so overwhelmed by the sudden development of the ever-present virus that antibodies on which the Wassermann reaction in all probability depends are not produced, and hence it happens that negative reaction may be obtained even in general paresis.

Dr. Charles R. Ball, St. Paul: From a close observation of the cases reported, and most of them I have had an opportunity to observe over a number of months, it is my impression that the injections of sodium nucleinate exert a much better influence on the symptoms than either salvarsan or mercury. The patient who received the largest number of salvarsan injections—eight in all—after a short period of improvement rapidly grew worse. The two patients, who at present, except for their somatic symptoms, are apparently well, have resumed their former occupations. Both of these patients I am still keeping under antispecific treatment during this period of remission. It will be interesting to know what the influence of antispecific treatment will be during this period.

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MEDICAL

TUMOR OF THE HYPOPHYSIS IN A CASE OF ACROMEGALY

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The importance to health of the integrity of the pituitary gland is now unquestioned. Experimentalists and clinicians are busy elucidating its physiology and pathology, in order that rules may be formulated for the diagnosis and treatment of its various disorders. Meanwhile surgeons are perfecting new practical methods of approach to the hidden gland at the base of the brain, thus robbing hypophysectomy of many of its dangers. The literature of the past few years has been enriched by a number of valuable articles and monographs on the theoretical and practical aspects of hypophyseal disorders, which must be consulted by those wishing to get a general view of the subject. My contribution is the report of a case of acromegaly with post-mortem findings, which I had the privilege of studying for about seven years. I shall attempt, if possible, to correlate the features of this case with the latter-day teachings on the hypophysis.

REPORT OF CASE

History.—The patient, a Canadian lake captain, 49 years old and unmarried, was first seen in September, 1903. Father died of some lung trouble at 71; the health history of other members on this side of the family is unknown. The mother, in good health at 79 years, stated that her parents died at 82 and 85 respectively; a sister had a "stroke" at 70 and died several years later; her other children are healthy and she

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never knew of any nervous diseases in her entire family. Birth and early physical and mental development of patient were normal. With the exception of an attack of tonsillitis when 2 years old and of two attacks of lumbago during adolescence, there were no complaints previous to the present trouble. He always had good habits; never smoked nor drank to excess. Though sexually moderate, he did not escape a mild case of gonorrhea and, later, a chancroid with bubo; but he never acquired syphilis. During his boyhood and early adolescence he was tall and slim. Figure 1 shows him at 35. At this time he was still within normal proportions with reference to height and weight. Soon after this picture was obtained, however, he began to gain in weight, and we discover in him the fully-developed acromegaly type at the age of 44 (Fig. 2).

Present Complaint.—At my first examination the family were agreed that the disease had not lasted over eight years. The patient placed much emphasis on two incidents which occurred shortly before the development of his symptoms, and he holds these responsible for his troubles. First, during a stormy voyage on the lake he was overtaken by fright and confusion of such nature that he was obliged to yield the command of the vessel to his first officer. The second incident which he regards as the principal cause of his disease was an infected vaccination wound on his left arm, from which he suffered in 1894. Shortly after the infection had subsided, he noticed peculiar nervous "spells," during which he held his breath and snored for a few seconds; there was said to have been some discoloration of the lips, but no frothing at the mouth during these attacks. Consciousness was never lost during any of the seizures. In the autumn of 1894 the attacks changed in character, not only motor, but also sensory and vasomotor phenomena occurring; added to the snoring and pause in respiration there developed "creepy," chilly feelings, followed by hot flushes and sweatings. Occurring at long intervals in the beginning, the "spells" became more frequent later, until toward 1897 major attacks of epilepsy also made their appearance. During a period of seven months, in 1895, the patient found himself in a condition of physical and mental inertia. He was somnolent, apathetic, could not recognize familiar objects and persons and was unable to attend his natural wants. Slowly he emerged from this lethargy; he began to take short walks in the open and to care once more for his person. At this time and subsequently an insignificant physical or mental effort was usually sufficient to bring on a state of exhaustion. In 1896 many nervous spells of the variety already described occurred, which were modified to the extent that each attack was either followed or initiated by a smacking of lips and trembling of face,

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usually accompanied by a lacrymose outcry of "Oh my, oh my!" In 1897 the first major attack of epilepsy was noticed; there was the short tonic, followed by a longer clonic convulsion, with frothing at the mouth, biting of the tongue, and complete relaxation of the vesical sphincter. Similar attacks continued to recur mostly during the early morning hours. They were always preceded by "creepy," chilly feelings in the fingers; often, however, the latter constituted the entire attack, which then terminated by yawning and profuse perspiration, but without convulsions. A prominent symptom was severe headaches, which have occurred at irregular intervals since the lake accident—for the patient the beginning of his disease. Very intense at first, the cephalalgias became less severe and less frequent toward the end.

Physical Examination.—Patient is about 6 feet tall, presents a clumsy posture and a gait betokening lack of energy. His face is coarse and large, the lower jaw massive and protruding beyond the upper; the lips and nose are thick, the nasolabial folds accentuated. The eyes are deeply set, eyebrows coarse, and supra-orbital arches prominent. While the lower portion of the forehead bulges out considerably, the upper end appears to be retreating; the malar bones are large. The fingers are sausage-shaped and the hands resemble a spade, all the tissues—bones, muscle, fat and skin—being excessively developed. The feet show similar excess in the growth of both soft and osseous structures. Kyphosis is marked in the cervicodorsal region. Both clavicles and the sternum are considerably enlarged. The genitals are undersized and the libido and sexual power are absent. Except for the heart, which shows slight tachycardia, the viscera are normal. The voice is low-pitched, the speech slow, gutteral and monotonous. The tongue, indented by the teeth, is extremely large and fills the mouth almost completely, thereby exciting an increased flow of saliva. The thyroid is not palpable.

At various times early in my observation I found in the urine phosphates, urates and traces of sugar, but no albumin. Somewhat later I failed to detect sugar, and discovered the phosphates only occasionally. Polyuria, polydipsia and polyphagia were symptoms more or less present during the entire course of the disease. A blood examination revealed nothing beyond a slight secondary anemia.

Nervous Examination.—There is no paralysis, tremor or ataxia. The tendon reflexes are reduced in the upper extremities; the knee-jerks are barely elicitable, the Achilles jerks absent. Babinski's toe phenomenon and Oppenheim's foot sign cannot be obtained. The sensory examination reveals negative findings throughout. The special senses show no

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anomalies. Hearing, taste and smell are normal. An examination for visual disturbances fails to disclose hemiopic or central vision disorders. As I could not prevail on the patient to visit an ophthalmologist for purposes of perimetric and ophthalmoscopic examination, I am compelled to use rough tests for the visual fields, which yield negative findings. Ophthalmoscopically, the optic nerve appears within the normal limits of health, neither congested nor too pale. That the patient has remained an inveterate newspaper reader almost to the end would lend support to the last statement. The pupils react to light and in accommodation, though rather sluggishly.

Mentality.—Forgetfulness, apathy, absence of mental concentration and a puerile state of mind are constant features. The patient is an indefatigable collector of newspaper clippings from the advertisements of quacks and charlatans, who appeal strongly to his enfeebled mind. On certain days he develops attacks of somnolency, which interrupt all serious occupation with the patient. He thinks a Roentgen-ray examination unnecessary and cannot be induced to go to the laboratory.

Subsequent History.—During the two years previous to his death the major epileptic attacks had become rare, but instead the *petit mal* seizures had become more frequent. In addition there developed great difficulty in swallowing, regular choking spells impeding the process of feeding. The troubles of deglutition diminished about three or four months before death, but the patient became more somnolent and weak; the memory was almost a blank; he was childish, confused and unable to answer simple questions. The patient died in coma July 27, 1910, at the age of 56, having been a sufferer from acromegaly for about fifteen years.

Necropsy.—Permission was granted to examine the brain, but none of the other organs. The important finding was a large tumor in the hypophyseal region, which had grown in all directions (Fig. 3). In its continuous growth, the neoplasm had destroyed the posterior clinoid processes and extended upward as well as downward. While the sphenoidal sinuses were also found infiltrated with tumor tissue, the principal mass grew laterally and upward. As a result of this upward extension the optic chiasm had become attenuated and flattened into a cup-shaped structure (Fig. 4), measuring 2 by 1.5 cm. Laterally the neoplasm grew from the sella toward the uncinate gyri, both of which were thus compressed, but especially the left one. Still further extending into the inferior horns of the lateral ventricles, the tumor produced a considerable degree of irritation hydrocephalus, which was more marked on the left side (Fig. 5). The irreg-

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ular mass, with its numerous protuberances occupying the entire interpeduncular space, was of a dirty-gray color and had but little cohesiveness; after a few days' hardening in formalin solution it began to fall apart. It measured approximately 6 cm. in its transverse, 4 cm. in the sagittal and 3 cm. in the longitudinal diameter.

Microscopically there are seen a few groups of deeply staining cells (chromophils) scattered among numerous others that stain but feebly (Figs. 6 and 7), (chromophobes), with colloid masses here and there. Part of the tumor shows irregular rows of cylindrical epithelium, having the typical appearance of secreting cells, with their darkly stained protoplasm to one side and the lighter portion to the other (Fig. 8), a picture resembling a secreting gland. The brain not having been especially prepared for the granular stain, the existence of granule cells could not be ascertained with certainty, though Dr. Dean Lewis, who kindly examined and stained for me some specimens from this tumor, believes that he recognized some of the cells on which he and Benda have laid so much stress. When I first examined the tumor the diagnosis of sarcoma was uppermost in my mind, because the growth consisted largely of round cells with little or no stroma. On consulting the literature, however, and especially Benda's and Lewis' writings, I came to the conclusion that the neoplasm belonged to the adenomatous variety of hypophyseal tumor.

Summary.—As a result of a psychic trauma or an infection a male patient at the age of 41 develops symptoms of acromegaly, accompanied by attacks of petit mal epilepsy; somewhat later there are added somnolency, cephalalgias and change of character. Though presenting the classic picture of acromegaly the patient never suffered from any of the usual visual disorders of this disease, such as hemiopia and central scotoma. In the course of time, however, attacks of major epilepsy and uncinate fits supervene and the patient dies about fifteen years after the beginning of the first symptoms, having lived the life of a vegetating automaton for many years. The post-mortem examination then reveals a large adenoma, which had pushed up the optic chiasm, extended into the lateral ventricles and incidentally caused a bilateral hydrocephalus, more marked on the left side.

CONCLUSION

The peculiar distribution and extent of the tumor may explain many of the symptoms in this case. The proliferation of glandular tissue leads one to suppose that there must also be a corresponding increase in the

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function of such tissue. Assuming with Benda,[1] Lewis[2] and Cushing[3] that the skeletal overgrowth of acromegaly is conditioned by a hypersecretion of the anterior portion of the hypophysis, we certainly had the structures from which such secretions might have been derived. As the tumor continued to grow, the hyperactivity was converted into hypoactivity—the chromophil gave way to chromophobe cells. Then it was that we discovered the existence of adiposity, myasthenia, and loss of sexual power. As may be observed in the microphotographs, the tumor masses consist almost entirely of chromophobe cells, while the chromophil cells—the real secreting structure—seem to have almost entirely disappeared. Apparently there was a stage of immoderate stimulation followed by one of exhaustion. This interpretation harmonizes with the modern teaching on the causation of acromegaly. While the theory of hypophyseal activity receives but scant support from my case, more can be said under the heading of neighborhood symptoms on which Cushing in his monograph puts great emphasis. We may recall that the patient developed *petit-mal* attacks at first, and, as the tumor continued to grow toward the uncinate gyri, there also appeared attacks which were accompanied by a smacking of the lips and movements as though the patient were tasting some unpleasant substance. These were probably *petit-mal* seizures belonging to the type which Hughling Jackson described long ago as uncinate fits—caused by the extension of the growth to the uncinate

1. Benda, C.: Beiträge z. normalen u. pathologischen Histologie d. menschlichen Hypophysis cerebri, Berl. klin. Wchnschr., 1900, xxxvii, 1205; Deutsch. med. Wchnschr., 1901, xxiii, 536; Pathologische Anatomie d. Hypophysis, Handb. d. path. Anatomie d. Nervensystems, 1904, p. 1418.

2. Lewis, D. D.: Hyperplasia of the Chromophil Cells of the Hypophysis as the Cause of Acromegaly, with Report of a Case, Bull. Johns Hopkins Hospital, 1905, p. 157; A Contribution to the Subject of Tumors of the Hypophysis, THE JOURNAL A. M. A., Sept. 17, 1910, p. 1002.

3 Cushing, H.: The Hypophysis Cerebri; Clinical Aspects of Hyperpituitarism and of Hypopituitarism, THE JOURNAL A. M. A., July 24, 1909, p. 249; The Functions of the Pituitary Body, Am. Jour. Med. Sc., 1910, xxxix, 473; The Pituitary Body and its Disorders, J. B. Lippincott Company. This excellent monograph contains the recent literature on the hypophysis.

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convolutions. The attacks of *grand-mal* epilepsy must also be considered as neighborhood symptoms. They were most probably caused by the varying fluid content in the ventricles compressing the brain against the skull, thus producing cortical irritation. To the invasion of the frontal lobe by the tumor must be attributed the mental changes, somnolency and puerile behavior—symptoms frequently noted in acromegaly. The absence of visual disturbances in my case can be explained from the appearance of the optic chiasm, as may be observed in the photograph. The chiasm appears thinned out, but widened; evidently the neoplasm compressed this structure very slowly, thus permitting gradual accommodation to pressure, while the tumor itself showed a tendency to extend laterally, rather than to grow in an upward direction.

A symptom which may throw some additional light, as every such case must, on the interrelationship existing between the various glands of internal secretion, was the complete absence of a thyroid gland in my patient. Possibly in taking up the work of the thyroid, the hypophysis continued to grow and functionate beyond the physiologic needs, with the resulting formation of a large adenoma.

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Fig. 1.—Patient at 35, still within the normal growth proportions.

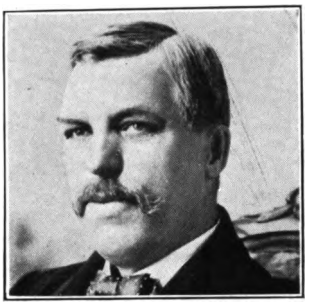

Fig. 2.—Patient at 44. Typical acromegaly appearance.

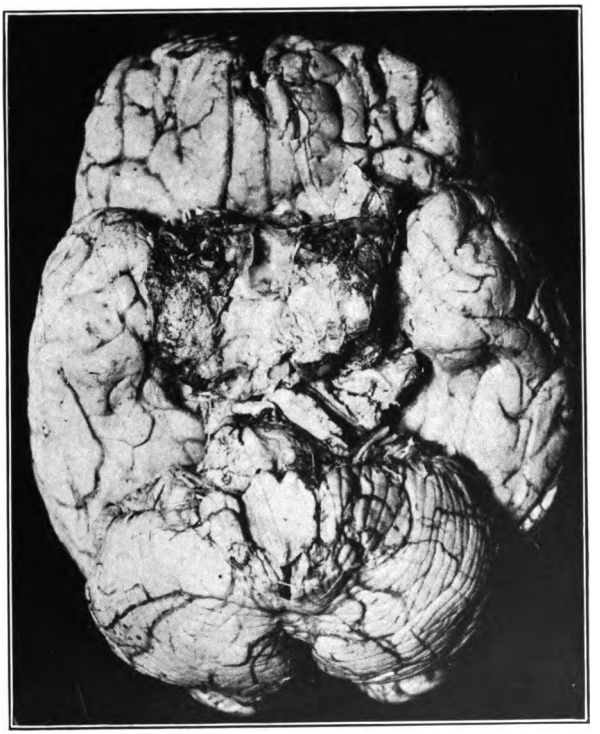

Fig. 3.—Hypophyseal tumor growing in all directions, more especially on the left side.

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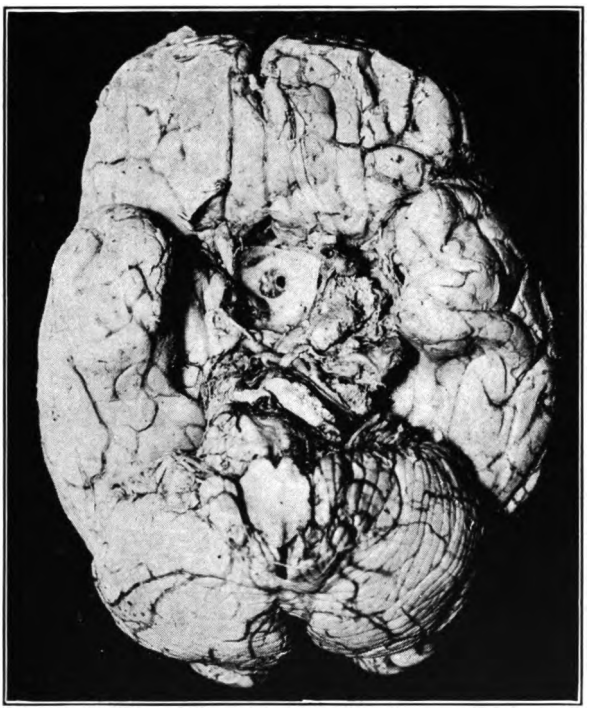

Fig. 4.—Optic chiasm, very much attenuated, surrounded on all sides by tumor mass.

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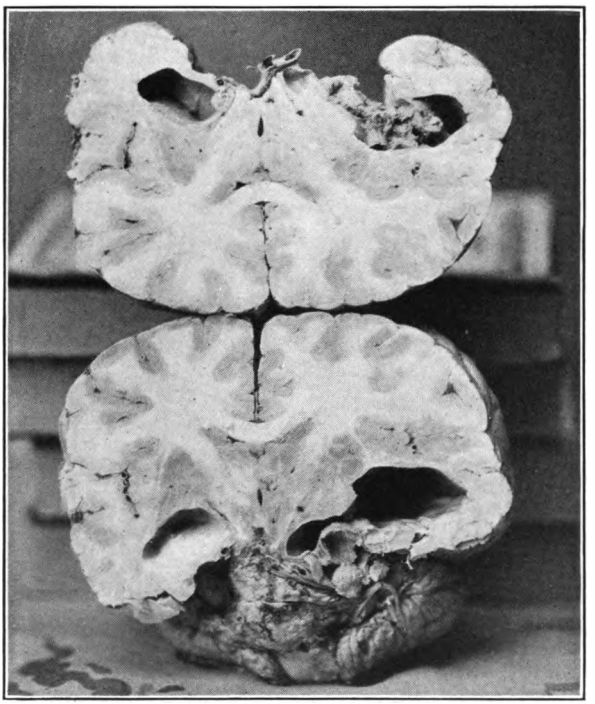

Fig. 5.—Bilateral hydrocephalus, more marked on left side. Infiltrating masses seen in left lateral ventricle.

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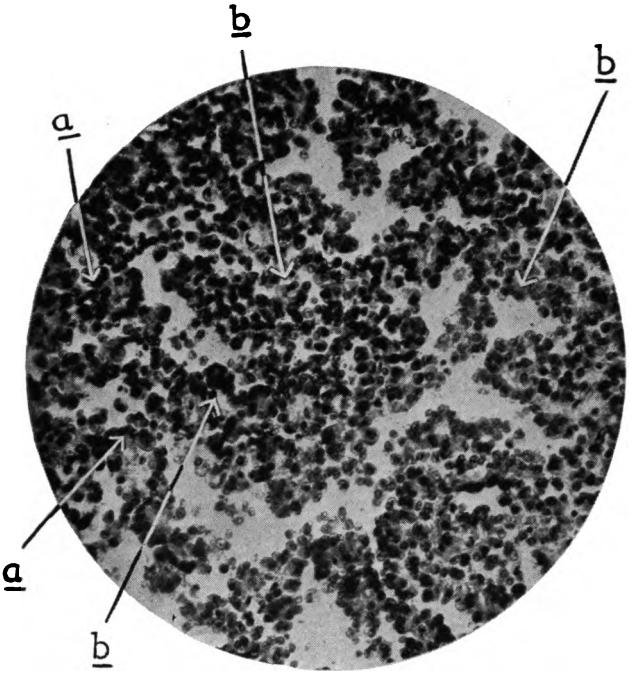

Fig. 6.—Magnified 180 diameters. Tumor consisting of (*a*) chromophobe cells, with few (*b*) chromophil cells interspersed.

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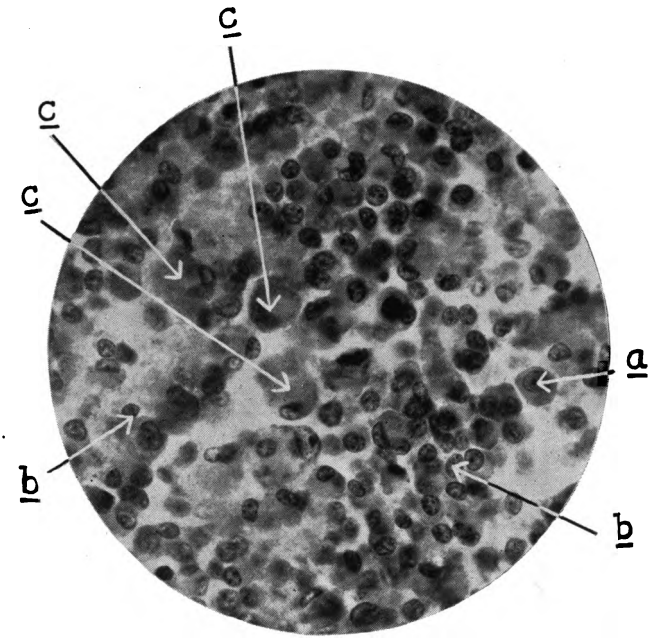

Fig. 7.—Magnified 450 diameters, showing (*a*) large and (*b*) small cells; also (*c*) colloid material.

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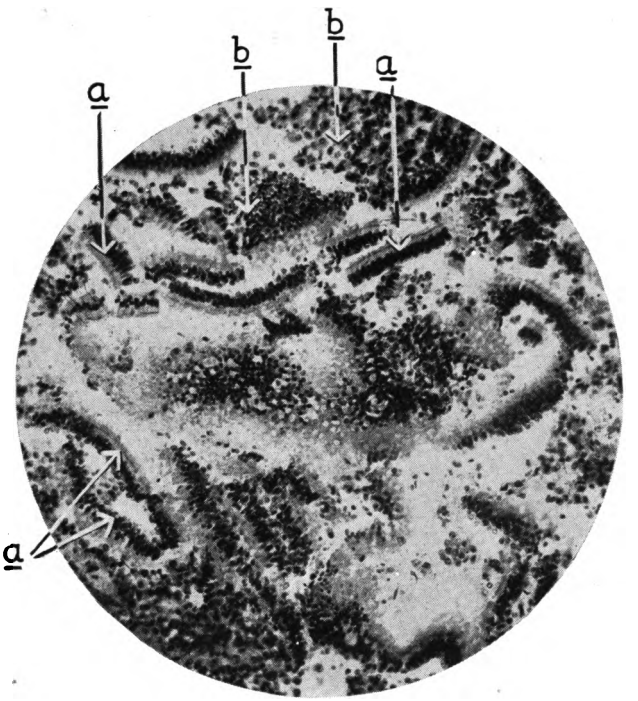

Fig. 8.—Magnified 110 diameters. (*a*) Irregular rows of cylindrical epithelium, resembling glandular structure; (*b*) scattered round cells like sarcoma.

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SYMPTOMATOLOGY OF MULTIPLE SCLEROSIS

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CHICAGO

Patient.—Mrs. F., aged 43, married twenty-two years, housekeeper, was seen in consultation with Dr. M. Barker. The father, aged 70, in fine health, does not drink, uses tobacco moderately and never had any severe, prolonged disease. The mother died of consumption when patient was an infant. Brothers and sisters are all healthy. Patient has five well-developed children; never had a miscarriage.

History.—The patient's birth was natural and she never had convulsions. Childhood was quite uneventful. School life was normal. Menstruation began early and still occurs regularly and painlessly, though rather profusely. The patient was well till fourteen years ago when she experienced an attack of paralysis, first on the left and then on the right side. She remembers a distinct tingling sensation traveling up the legs and arms. No disturbance of consciousness occurred with this attack of paralysis. There was some difficulty in controlling the bowels and she thinks she had some trouble retaining the urine. All this lasted about ten days, after which she seemed to recover except for the left hemiplegia which disappeared only in part. Some months after this the right side became gradually paretic again, but was never so bad as the left. The patient's mentality being somewhat below par, her laughter at times being most impulsive and immoderate and her husband not remembering well his wife's earlier condition, it was not possible to learn more of the patient's history.

Examination.—The gait was typically that of a mildly spastic paraplegia. The knee-jerks were highly exaggerated, and all of the usual symptoms of pyramidal involvement, such as the ankle-clonus, the Babinski, Gordon, Oppenheim and Chaddock signs were all easily demonstrated. The abdominal reflex was absent. Incoordination and intention tremor were most obvious in the hand and arm movements. Tactile sensations

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seemed to be well preserved everywhere except in and about the right hypogastrium. No analgesia could be detected and the thermic sense was only now and then slightly confused. Nystagmus, particularly on extreme lateral tension to the left, was marked. Only the slightest degree of speech disturbance could be detected.

An examination of the eyes proved negative except for a slight temporal pallor of the disks. At times there was a little difficulty in swallowing and a sense of choking. The mentality was hysteroid in character, and impulsive and immoderate laughing occurred. A Wassermann blood-test proved to be negative. Diagnosis: Typical disseminated sclerosis.

It is not my purpose to describe, or even to enumerate, all the symptoms that have been credited to multiple sclerosis. Last year,[1] I attempted to define sharply the pathology of the disease in order to distinguish it from a mere disseminated encephalomyelitis and thus to account for the wide diversity of opinion among equally competent authorities as to the frequency of multiple sclerosis. It is my desire to attempt the same thing this year in connection with the symptomatology.

It is an easy matter to diagnose multiple sclerosis clinically when the Charcot triad of symptoms is present. Nosology long ago determined that when the so-called intentional tremor, nystagmus and scanning speech, with some evidence of more or less involvement of the pyramidal tracts were present the case should be acknowledged as one of insular sclerosis. Such clear-cut cases, however, as every clinician must admit, are exceedingly rare. Even cases in which one or two of these cardinal symptoms are characteristically pronounced are rare enough. Therefore those conservative diagnosticians who hold rigidly to the Charcot picture of the disease will find their list of cases small, and their view will be that multiple sclerosis is an exceedingly uncommon affection. In this view they will receive some support from the fact that occasionally these cardinal symptoms have been observed in cases which on necropsy

1. Mettler, L. Harrison: Multiple Sclerosis, THE JOURNAL A. M. A., Nov. 2, 1912, p. 1607.

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proved to be entirely different maladies, as for example the pseudosclerosis and diffuse scleroses, malarial intoxication, cerebrospinal syphilis, and dementia paralytica.

The query here forcibly intrudes itself, What are we to do, go on making erroneous diagnoses in calling all sorts of maladies insular sclerosis because forsooth they happen to present a nystagmus with a spastic gait, a disturbed speech or a peculiar tremor; or are we to limit our diagnosis of insular sclerosis to the strict Charcot picture and call all the others that merely suggest a disseminated lesion by some other name; or admit, if in the last instance no other name than multiple sclerosis can be applied, that no positive diagnosis is possible? As it seems to me, this is the clinical problem to-day in regard to multiple sclerosis; and until some agreement is obtained in regard to these questions, all further discussion of the frequency of the disease, based on clinical statistics, is a waste of time and energy. In a word, the literature of multiple sclerosis to-day declares as plainly as language can that every diagnosis of multiple sclerosis, in the absence of the typical Charcot picture, is but a guess, and here as well as in the cases that present a modified or atypical Charcot picture the positive diagnosis can be made only at the post-mortem table.

That the foregoing criticism is not unduly harsh, let me illustrate by the following report of a case from a well-known clinician, typical of the literature of multiple sclerosis:

The patient, a girl aged 19, after a week's sickness vomited, had pains in the abdomen and was unable to walk. She was bright and cheerful and the first diagnosis was that of hysteria. In a few days the incontinence of urine and feces awakened the suspicion of an organic basis for the symptoms. Finally the diagnosis of disseminated sclerosis was made on the following summary of symptoms (here I quote from a prominent medical journal of London):

1. Patient in bed, fairly cheerful, but disinclined to speak.

2. Inability to stand, although both legs are moved freely in bed.

3. Reflexes exaggerated, no tremor, and apparently no anesthesia.

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4. Dilatation of the left pupil, with left ptosis, and flattening of the left side of face.
5. Incontinence of urine and feces.
6. Occasional purposeless vomiting; apparently no headache.

The diagnosis vibrated between cerebral tumor in a silent area, tuberculous meningitis and disseminated sclerosis. It finally settled on the last-named, though why I do not see. The reporter held that this diagnosis was established at necropsy, when on "minute examination of the hardened brain" there appeared "numerous areas of degeneration throughout, their shape, size and distribution being very irregular. There was no shrinking or bulging of the cord at the sites of the degenerative areas."

All of these symptoms and pathologic findings fail, in my judgment, to establish the diagnosis of multiple sclerosis. A general infective process with a disseminated lesion does not necessarily mean multiple sclerosis. No wonder some men find the malady so common while others find it so rare!

The danger of calling every case of encephalomyelitis, the lesion of which happens to be disseminated, multiple sclerosis lies in the fact that many forms of curable disease will be regarded as progressive and hopeless. Not a few of the infections of the nervous system reveal a scattered condition of the lesion, and if such a pathologic condition should happen to exhibit a clinical picture so strongly similar to that of multiple sclerosis as to lead to the latter diagnosis, the real nature and possible curability of the disease are bound to be overlooked. Even more emphatically will this be the case if we cut loose entirely from the classical symptomatology of the disease and call cases of disseminated sclerosis those which do not present the tremor, nystagmus, speech scansion or spastic condition of the limbs.

Clinically the problem narrows itself to the question as to whether the classical picture of Charcot is to be regarded as a mere syndrome, a localization picture only, or as a sign of a distinct disease entity. If the former,

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then it would be better to drop the term "multiple sclerosis" entirely since we have come to regard that as a progressive malady and incurable, and to emphasize in our diagnosis merely the infectious process underlying the particular case in hand and the disseminated character of its lesion.

If the latter view is to prevail, then it would be wiser to adhere strictly to the Charcot picture of multiple sclerosis and to endeavor to differentiate its symptomatology from that of a mere disseminated encephalomyelitis of infectious origin. To illustrate: It would be better to make the diagnosis dementia paralytica, though its symptoms may in some respect resemble those of multiple sclerosis, than to establish the diagnosis of the latter, as has not infrequently been done and long after, even after death, discover that the malady was syphilis of the central nervous system with, in the case in hand, a more or less disseminated lesion. In other words, there is a practical advantage, and even safety, in clinging more rigidly than has been the tendency of late to the older and more strict definition of multiple sclerosis. The farther we drift away from that anchorage the wider will the sea of confusion become and the larger will the number be of erroneous diagnoses.

To accomplish the result which I have just indicated, namely, the more accurate diagnosis of genuine multiple sclerosis, two things will be attempted, admittedly difficult as they are. First, the positive and characteristic symptomatology of typical multiple sclerosis, so far as the cases that have been verified by necropsy warrant it, will be outlined; and secondly, the symptomatology of a mere disseminated encephalomyelitis, with whatever shades of difference there may be between it and the previously indicated symptomatology of multiple sclerosis, will be described.

Of course no one symptom will establish the diagnosis of any disease. Two or more, if one includes a cardinal symptom, may. The positiveness of the diagnosis

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increases with the number of cardinal symptoms included in the general picture. In multiple sclerosis, of the Charcot type, perhaps the two most distinctive symptoms are optic atrophy and more or less spastic paraplegia with its usual accompaniments in the way of heightened knee-jerks, Babinski phenomena, clonus, etc. Next in importance, and of course when added to these confirming the diagnosis almost absolutely, is the tremor, whether it be manifested as an intention tremor in the hands, a scanning in the speech or a nystagmoid movement in the eyeballs.

All of these symptoms are so well known that I shall not presume to waste time describing them. I desire in naming them in this order merely to point out that after leaving this group of manifestations we have absolutely nothing whereon to base a positive diagnosis of multiple sclerosis or any form of the disease. With one or more of these distinctive manifestations are often seen a whole host of other symptoms, psychologic and physiologic, which may suggest the diagnosis of multiple sclerosis but which in a goodly number of cases will be found to belong to some other malady with a disseminated lesion. It is just at this point that the erroneous diagnosis of multiple sclerosis is so often made, as shown by post-mortem examination. It is a risky business to diagnose multiple sclerosis on these non-cardinal symptoms when one has in the picture only one of the cardinal signs. It is nothing but a gratuitous guess and no diagnosis at all to call the case one of multiple sclerosis when the signs present include all of the non-cardinal signs but not one of the typical manifestations. Nevertheless, a survey of the literature of multiple sclerosis shows that just that sort of diagnoses are being made every day. The uncertainty and inaccuracy of these diagnoses obviously render such literature quite worthless for scientific purposes. Among the non-cardinal symptoms, by way of illustration, I may enumerate the slight mental changes, the fugitive pains, slight

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paresthesias and other variable sensory disturbances, the apoplectic and epileptiform seizures, the bladder and bowel irregularities, the pupillary inequalities and the bulbar manifestations. All of these symptoms appear more frequently in other maladies than in multiple sclerosis. In the latter they are incidental rather than diagnostic.

Peterson names six cardinal symptoms of multiple sclerosis: muscular weakness sometimes with cranial palsies, intention tremor, exaggeration of the knee-jerks and wrist-jerks with possibly jaw-jerk and ankle-clonus, nystagmus, speech disorder and various signs of nervous disturbance such as vertigo, headache and epileptiform and apoplectiform seizures with mental dulness. With the exception of the last-named group, this list corresponds with the cardinal symptoms which I have already enumerated. The last group, or various signs of nervous disturbance, is not distinctive of any special nervous malady but occurs more or less in all. Therefore I cannot see how they are cardinal in multiple sclerosis or help us *per se* in diagnosing this disease. In fact the same is true of the muscular weakness, the general spastic paraplegic signs and the nystagmus. All of these occur frequently in other troubles. If anything is characteristic about them when they appear in disseminated sclerosis, it is the fact that they never go on to completion. The muscular weakness never becomes actual paralysis. The nystagmus is never so typical as in labyrinthine disease. The exaggeration of the reflexes is never of the highest degree, as seen in lateral sclerosis of the cord. It is this incompleteness of the manifestations that is the cardinal feature of the disease rather than the manifestations themselves. This leaves, therefore, as the characteristic symptoms of disseminated sclerosis, only the peculiar optic atrophy, the intention tremor, the speech disorder and the incompleteness of the spastic paraplegia. To these some authorities have added the loss of the abdominal reflex, though the uni-

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formity of this has not been such, in my judgment, as to dignify it with the adjective cardinal.

It is on the cardinal symptoms that multiple sclerosis assumes a definite nosologic status. Without one or more of the cardinal symptoms, multiple sclerosis as such cannot be recognized clinically. A curious problem obtrudes itself at this point. How are we to explain such a uniformity of symptomatology with such a wide-spread and haphazard lesion as multiple sclerosis presents? Why, with such a lesion, are the motor manifestations so frequent, so uniform and so distinctive, while the sensory are so indefinite and insignificant? Why are the vision and the optic nerve so often affected while the smell, taste and hearing are so rarely involved? How does it happen that such a scattered and haphazard lesion produces such pronounced external phenomena and yet such relatively slight visceral disturbance? And finally, why are some of the symptoms so permanent while others, though almost cardinal, are so transitory? Can it be that pathologically multiple sclerosis is one thing, namely, a wide-spread lesion, but clinically is quite another thing, a mere localization syndrome representing a part of the lesion?

In some respects it looks as if our classical conception of multiple sclerosis were limited to the disturbance of the apparatus of equilibrium and coordination. This is suggested by the cerebellospastic gait, the nystagmus, the speech scansion and the intention tremor. Cases of disseminated myelitis, involving the cord, medulla, pons and brain are not entirely unlike multiple sclerosis in their symptomatology, but there are usually other symptoms that help one to differentiate the former from the latter. There is a slightly different etiology between the two diseases as, for instance, the presence of a more or less neuropathic heredity in the sclerosis. On careful and prolonged observation the age at which multiple sclerosis is prone to appear, the sharper and more fixed character of its essential symptomatology, its general

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course and whole complexion are somewhat different from that of disseminated myelitis. The differentiation is difficult—extremely difficult—but it is not impossible. In the broadest view disseminated myelitis has many features about it that do not appear in multiple sclerosis. When, however, the myelitic lesions happen to entrench on and involve the functions of the general apparatus of equilibration, then its features may assume the characteristics of those of multiple sclerosis.

Disseminated myelitis is characterized by extensive paralyses with atrophy and disturbances of sensation, even more so than is ordinary transverse myelitis. In both they are decidedly more marked than they are in disseminated sclerosis. In the case of disseminated myelitis reported by Taylor and quoted by Starr, there were pains, incontinence of urine, marked weakness and numbness, and finally paralysis of the legs. There were high temperature, dyspnea, complete paraplegia, cystitis, retention of urine and tympanitic and resistive abdomen.

The point is that if we add the symptomatology of a disseminated encephalitis to the symptomatology of disseminated myelitis, even though, as Starr says, "disseminated encephalitis of infectious origin is the starting-point of many cases of so-called multiple sclerosis," we never find exactly the characteristic picture that we do in this so-called multiple sclerosis. There must be some element that marks the two sets of lesions one from the other. In the etiology there are the more marked hereditary and neuropathic preliminaries to the outbreak of multiple sclerosis; and in the symptomatology is that trait that clearly suggests involvement of the cerebellum or at least the cerebellar general apparatus, and all its connections, that preside over the function of equilibrium and coordination; also the age (early) of multiple sclerosis and the relative absence of syphilis.

Until there is a stronger basis for the view that disseminated encephalomyelitis is the same as disseminated

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sclerosis, we are justified in holding that the latter disease is a most rare affection, and in discarding from our statistics those reported cases that are called atypical and are yet unverified by a careful and rigid postmortem examination.

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ABSTRACT OF DISCUSSION

DR. C. D. CAMP, Ann Arbor, Mich.: I am surprised that Dr. Mettler did not mention the examination of the spinal fluid. I believe that this examination is important for differentiating multiple sclerosis from some of the conditions which are frequently mistaken for it. I think that Dr. Mettler referred to two cases reported by Dr. William G. Spiller and myself, in which the diagnosis was multiple sclerosis; in fact, the cases had been demonstrated to students as being typical of multiple sclerosis in every respect, but at necropsy they were found to be cases of syphilitic meningomyelitis. The title of this paper was "The Clinical Resemblance of Cerebrospinal Syphilis to Disseminated Sclerosis" (*American Journal of the Medical Sciences*, June, 1907). Such an error would not have occurred at the present day, I am sure, because the examination of the spinal fluid would have shown the correct diagnosis.

DR. JULIUS GRINKER, Chicago: It is true, as Dr. Mettler has stated, that we cannot always clinically differentiate encephalomyelitis from multiple sclerosis. It appears to me, however, that the only way in which we are at present able to diagnose multiple sclerosis is by excluding every other possible condition capable of producing the symptom-complex. When we discover a condition, the result of multiple foci, which is not syphilitic and not due to vascular disease, we have to think of multiple sclerosis regardless of whether or not we have Charcot's triad or any of the other symptoms of insular sclerosis. Nonne, in his book on syphilis of the nervous system, says that he is now convinced that he was in error when he believed that multiple sclerosis may give a positive Wassermann reaction. The only other condition that might occasionally give rise to difficulties in differentiation is brain tumor. Not long ago a patient was sent to me who presented symptoms of brain tumor. The patient had Jacksonian fits but no optic neuritis and Babinski sign on one side with only exaggerated reflexes on the other. In the absence of other symptoms, I assumed that the patient had a cortical tumor, something in the nature of a cyst. An operation was performed, but no tumor was found. Instead a large patch of multiple sclerotic tissue was discovered.

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Several foci together had given rise to the symptoms of a brain tumor. The patient died and multiple sclerotic foci were found in various other parts of the central nervous system. Such errors we seem to have no means of preventing. Otherwise I believe Dr. Mettler's point of view is not tenable, for, if I understand him correctly, he states in substance that because it is often impossible clinically, from the symptoms alone, to diagnose multiple sclerosis we should not diagnose it at all. I believe the disease to be more common than he states, and if we follow the rule of diagnosis by exclusion—by which I mean the seeking out of every possible condition and excluding it definitely, not only clinically, but also by means of all the new laboratory tests—we shall be able, in most cases, to diagnose multiple sclerosis.

Dr. D. S. Booth, St. Louis: It is only a few months since I had occasion to look up the literature on this subject, on account of a clinical case which came under my care, the result of which was the discovery that atypical cases much resemble several other diseases. The patient was a girl about 15 years old who had probably had symptoms of one kind or another all her life, so that the case brought out very prominently the possibility of Friedreich's ataxia. I found in the literature that this condition at times resembles atypical multiple sclerosis. With great difficulty I arrived at a conclusion and, even when I finally decided on a diagnosis of multiple sclerosis, I stated to the medical class before which I presented the case that there was a possibility of an error since I had found that eminent clinicians had erred in differentiating this disease from several other diseases which it may closely simulate.

Dr. Charles W. Hitchcock, Detroit: If I understood Dr. Mettler correctly, he restricts the term "multiple sclerosis" to a quite typical syndrome, and yet he has pointed out adequate reasons from the pathology of the disease why multiple sclerosis should be distinctly atypical. It does not seem to me that we should expect this disease to present typical symptoms and typical development; nor should we shrink from the diagnosis if it be difficult and somewhat uncertain, as we do not shrink from the diagnosis of syphilis of the nervous system. The varying signs of sclerosis, varying from the location and size of the pathologic lesions, would seem to indicate an atypical disease; therefore, I do not see why we should reserve the term "multiple sclerosis" for the typical syndrome if we have in mind the pathology of anything which develops atypically. It is a risky business, perhaps, for me to speak without having had any pathologic proof of some of my diagnoses, but I have had a number of cases, diagnosis of which I have been able to reach by such exclusion methods as Dr. Grinker has mentioned. These cases have been extremely atypical. I recall three in the past year,

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in each of which I was forced to a diagnosis of multiple sclerosis, but no one of which conformed to any cardinal type of the disease.

Dr. L. Harrison Mettler, Chicago: In reply to Dr. Camp I will confess that for the moment I did forget what everybody nowadays knows, that a cytologic and serologic examination of the spinal fluid will determine the presence of the syphilitic infection. There may be other infections present, however, which such an examination will not aid us in establishing. I agree, in the main, with Drs. Grinker and Hitchcock that multiple sclerosis can be diagnosed by exclusion; yet a diagnosis by exclusion is always largely a makeshift, and, although we are sometimes forced to do this, it is always a most desirable thing to avoid making a diagnosis in this way. Too often multiple sclerosis, like hysteria and neurasthenia, is made a sort of waste-basket into which is thrown everything which by exclusion cannot be diagnosed as some other malady. The literature is full of these uncertain diagnoses of multiple sclerosis, and this is the reason that I have presented this paper on the symptomatology of the disease. These diagnoses by exclusion, of which the literature is so full, are in most cases no diagnoses at all, or at least are so unreliable as to render the reported cases practically worthless in forming statistics as to the frequency of the disease. A frank confession that no diagnosis is possible is usually safer and more scientific than the average diagnosis by exclusion.

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TWO CASES OF CIRCULATORY DISTURBANCE OF THE BRAIN

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AND
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We thought it might be of interest to report to this section two quite rare and extremely interesting cases of circulatory disturbance of the brain.

CASE 1.—The first case is, in all probability, a thrombosis of the posterior-inferior cerebellar artery. It was referred to us by Dr. D. C. Jones.

History.—The patient is a man, aged 47, married, traveling salesman. He uses alcohol in moderation and tobacco in excess. Personal history is negative, except that the patient had gonorrhea at the age of 22; he denies lues. For the past four years he has had bilateral occipital headaches, more marked on the right side and more severe in the recumbent posture. Family history is negative.

Present Trouble.—The present disease began July 12, 1912. While out walking, the patient suddenly developed an attack of dizziness and staggering, so marked that he had to lean against a building to keep from falling. This lasted about ten minutes and was accompanied by diplopia and dull occipital headache. The diplopia disappeared but headache continued for three days, then stopped for one day and patient felt well enough to go fishing. The same evening he felt feverish, but slept well all night. On arising the next morning, he staggered so that he could hardly walk, and had a tendency to fall toward the right side. While washing, he noticed that the cold water felt warm to his right face and left hand. His voice was hoarse; the diplopia had returned, and he had difficulty in swallowing. During the past week he has had a slight tremor of the left arm and chin. Occipital headache was so severe in the recumbent position that patient had to sleep on four or five pillows. This headache disappeared almost entirely in about fifteen minutes after patient stood up. Physical examination was normal.

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Neurologic Examination.—Both pupils were equal and reacted to light and accommodation. The fundus of the eyes was normal. Fields of vision were normal; there was no nystagmus. Mild paresis of the right internal rectus muscle was present. Corneal reflex on right side was lost, on left side normal; there was slight drooping of right upper eyelid. On the right side of the face, over the area supplied by the first and second branches of the trigeminal nerve, pain and temperature sense were impaired, while tactile sense was normal. Over the remainder of the face all sensations were normal. Taste sense was normal. There was mild paresis of right facial muscles. Hearing was normal. There was mild paresis of right soft palate and right vocal cord. Patient's voice was hoarse and he swallowed with difficulty. There was no paralysis of the tongue muscles. Romberg was marked, patient being unable to stand alone, his eyes open and legs spread. The examination of the extremities showed the following: muscular strength normal in upper extremities. No ataxia; reflexes normal. In the lower extremities there was no loss of muscular strength. Knee-jerks and Achilles tendon reflexes normal and equal. Babinski and Oppenheim present on right side, absent on left side. No ankle-clonus. Tactile sense normal throughout, while pain and temperature sense were markedly impaired over left arm and left leg and left half of the trunk except the neck region. Deep sensations normal, no astereognosis. All superficial reflexes normal. The Wassermann in the blood was negative. Blood-pressure 138 mm. Hg. Urine normal, except for presence of sugar, 5.4 per cent. Patient voids 68 ounces of urine in twenty-four hours.

Progress of Case.—With rest in bed and increasing doses of potassium iodid he gradually improved. Diplopia and headache disappeared entirely in a few weeks. Sensation improved except over the right face, where it is still impaired. There is also a constant feeling of tightness in the throat. Improvement continued for six months, but on Dec. 7, 1912, patient had a similar mild attack, lasting about ten days and accompanied by occipital headache, hoarseness of speech and difficulty in swallowing. Since then he has made favorable progress and for the past two months has been attending to business regularly. The sugar in the urine still remains and varies between 1 and 8 per cent., and the daily amount of urine excreted is about 120 ounces. The sugar output is not affected by diet, and the patient shows no other symptoms of diabetes. Two years ago he was examined by an insurance company and at that time the urine was normal.

Summary.—Reviewing the case briefly, we have the following: Severe occipital headaches for the past four years, more marked in the recumbent posture; sudden attack of vertigo and diplopia, followed five days later by an attack of

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staggering, hoarseness, difficulty in swallowing and impaired pain and temperature sense over the right face and left extremities. A negative Wassermann in the blood, a blood-pressure of 138 mm. Hg and urine loaded with sugar. Patient had a mild similar attack six months later. With rest in bed and potassium iodid he made a practical recovery except for sensory involvement of right face, a feeling of tightness in the throat, and persistent glycosuria.

The localization is not difficult; the involvement of the vocal cord, the difficulty in swallowing and the disturbance in speech point to a lesion in the medulla, at about the level of the ambiguus and the glossopharyngeal nuclei. Through this region also passes the spinal portion of the fifth nerve. According to Schlesinger, pathologic-anatomic studies have shown that the arrangement of the fibers from the centers of the fifth nerve are such that those in the distal portion supply mainly the first and second branch, while the proximal portion of the spinal trigeminal root supplies the third branch and controls the sensations of the lower jaw and taste. In our case, the third branch was not affected at all; hence, the lesion could not have extended into this region.

Henschen, in reporting a case similar to ours, remarks that tactile fibers ascend in the posterior columns and are next to the raphe. Therefore, they usually escape in these bulbar lesions. He also states that he has demonstrated in several cases that destruction of the fillet causes loss of tactile as well as of pain and temperature sensations; therefore, the latter fibers enter the fillet more proximally than the medulla oblongata. According to Müller, the spinal tracts for pain and temperature sense decussate soon after their entrance into the posterior horns and ascend by way of the spino-tactile and spinothalamic tracts. This accounts for the loss of pain and temperature sense on the side opposite the lesion. The marked ataxia and the tendency to fall to the right point to an involvement of the right cerebello-olivary tract and the restiform body. The dysarthria is due to involvement of the hypoglossus

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nucleus and must have been slight on account of its transient character. The paresis of the right internal rectus and the involvement of the muscles of the right face point to a lesion of the abducens and facial nuclei. This must have been of a very mild degree, however, for both the diplopia and the facial paralysis entirely disappeared in a few days. The occipital headaches of four years' duration might suggest a brain tumor, but the subsequent improvement and the absence of signs of intracranial pressure readily eliminate this probability. The change in the severity of the headaches, in different position (that is, more marked in the recumbent posture and milder in the erect), points to a circulatory disturbance, probably of arteriosclerotic nature.

E. Schwarz reported a similar condition in his case. Although our patient had a normal blood-pressure and no signs of arteriosclerosis, one must not forget that isolated arteriosclerosis of the cerebral blood-vessels is quite common. Bonhoeffer, in a recent article, discusses this at length. Both Mann and Marburg have spoken of the occurrence of central pains, especially in the region supplied by the trigeminal and occipital nerves in lesions of the pons or medulla, and believe that they are due to a variation in the filling of the arteriosclerotic blood-vessels. According to Oppenheim, neuralgic pain of several years' duration may be a prodromal symptom of a subsequent thrombosis. These facts might help to explain the headache of four years' duration in our patient. In Bonhoeffer's case the occipital pains ceased soon after thrombosis of the artery had taken place, this being similar to what occurred in our case. According to Duret and Senator, the left vertebral artery is usually the seat of the obstruction, possibly because its course is more in the direction of the subclavian artery and also because it has a greater blood-pressure.

Wallenberg, after carefully studying the course of the injected vertebral blood-vessels, reports the following:

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Each of these arteries gives origin about 2 cm. below their union to the posterior-inferior cerebellar artery and higher to the anterior spinal artery. The latter artery passing downward enters the raphe and supplies the interolivary bundle, the posterior longitudinal bundle, the hypoglossus nucleus and other nuclei below the floor of the fourth ventricle. The pyramids are usually nourished by the vertebral arteries. Branches from the posterior-inferior cerebellar artery or the vertebral artery supply the lateral portion of the medulla oblongata, while the posterior-inferior cerebellar artery also supplies the restiform body.

We are at a loss to account for the persistent and marked glycosuria. It is quite possible that we are dealing with a simple case of diabetes. But the only symptoms pointing to this are the polyuria and glycosuria. Strümpell states that quite occasionally one finds a marked glycosuria following a brain hemorrhage. Müller reports a case of thrombosis of the posterior-inferior cerebellar artery accompanied by transient glycosuria and albuminuria. According to Morris, from the posterior-inferior cerebellar artery, branches are given occasionally to supply the chorioid plexus and the fourth ventricle. Could it be possible that the floor of the fourth ventricle has been irritated sufficiently to produce this glycosuria?

It would have been of interest to study the cerebrospinal fluid at the time of the first examination, but conditions were so serious that it did not seem justifiable to advise a lumbar puncture.

The brain circulation, according to Duret and Huebner, can be divided into two groups, the system of the cortical arteries and that of the central arteries. The cortical system anastomoses freely with the blood-vessels of the pia, and also with itself. After the blood-vessels have entered the cortex anastomosis no longer occurs (Beevor). The central arteries have been considered strictly end-arteries, but according to Hirsch this will

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bear further investigation. Kolisko has shown that the chorioid arteries anastomose with the lenticulo-optic and lenticulostriate arteries, while Wallenberg, through careful studies of the circulation of the medulla, has pointed out that anastomosis occurs much more extensively in the arteries of this region than was formerly supposed. Frequently the symptoms following disturbed brain circulation are entirely out of proportion to the slight extent of the pathologic lesion found. This probably is due to reestablished circulation of the anastomosing blood-vessels. Thomas reports a case of thrombosis of the posterior-inferior cerebellar artery in which at autopsy no necrosis was found in the medulla, and he explains its absence by the rich collateral circulation.

CASE 2.—The second case that we have to report is a ruptured brain aneurysm. The case was referred to us by Dr. George Earl.

History.—Patient is a woman, 44 years old. Family history is negative. Patient suffered frequently as a child from severe headaches, which were entirely relieved by wearing glasses. At 32 she had a fall from a swing and was unconscious for a short time. For the past three years she had been in the best of health. After eating her mid-day dinner, Jan. 26, 1913, she went to her room and about four hours later some one heard a commotion and found that the patient had fallen down stairs. She had vomited and was semiconscious. With help she walked upstairs, but soon developed a restless delirious state. This condition lasted until her death, six hours later.

Examination.—The patient was cyanotic; her breathing was stertorous. The reflexes were normal; there seemed to be a slight flaccid paralysis of the left lower extremity. The background of the eyes was normal. There was no involvement of the cranial nerves. The physical examination revealed nothing definite; the blood-pressure was 90 mm. Hg, and the urine was normal.

We suspected a pachymeningitis hemorrhagica or an acute menigitis, and advised a lumbar puncture. This was done and apparently pure blood under pressure escaped from the needle. During the process of the lumbar puncture, the patient suddenly died.

The centrifuged spinal fluid gave a heavy reddish precipitate, leaving the supernatant fluid a yellowish color. The

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examination of the precipitate showed many normal red blood-cells, a few polymorphonuclear leukocytes and a few lymphocytes. No bacteria were found. This aided us in making a diagnosis of a recent hemorrhage into the subarachnoid space.

Post-Mortem.—This was performed seventy-two hours later. There was nothing particular to note in the organs of the thorax and abdomen except an hour-glass stomach. The skull was of normal thickness. The dura was nowhere adherent. There was no indication of a fracture of the skull. When the dura was opened the brain was found bathed in blood. Blood-clots were seen, especially at the base of the brain. The blood was readily removed by washing and a small ruptured aneurysm, about the size of a hazelnut, was found near the bifurcation of the left posterior cerebral and the posterior communicating arteries. It is of interest to note that there were no previous symptoms indicative of an intracranial lesion.

According to Krey, rupture of a brain aneurysm occasionally occurs without any prodromal symptoms, but usually headache of a paroxysmal nature and pain in the neck and occipital region are present for some time previously. It is quite common to have involvement of some of the cranial nerves, especially the oculomotor and the optic. Krey bases his observations on 108 cases from the literature and his own material. Blowing murmurs on the skull, synchronous with the pulse and disappearing by compression of the carotid arteries, are very characteristic. But a similar blowing murmur may occur in angiosarcomas, in tumors of the corpora quadrigemina pressing on the vena magna galeni, in internal hydrocephalus, and occasionally in normal healthy children. Sometimes brain aneurysms are accidentally found at post-mortem. During life they frequently produce no symptoms up to the time of rupture and then death usually results. According to Lebert and Hey this occurs in over 50 per cent. of all cases. They usually occur at the base near the bifurcation of the larger cerebral blood-vessels; the sylvian, the basilar and the vertebral arteries are most frequently involved. The aneurysm is usually sacculated, but the spindle-shaped variety also occurs. The size varies from that

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of a pea to a hen's egg. The arteries on the left side are more frequently involved than those on the right. Although brain aneurysms are quite rare, they occur more frequently than in any other arteries of the body. They may occur single or multiple. Nothnagel reports a case of luetic endarteritis in which three brain aneurysms were found, two of which had ruptured at the same time. Clinically it is often quite difficult to differentiate this condition from a pachymeningitis or an acute meningitis. The examination of the cerebrospinal fluid is a valuable diagnostic aid, for in almost no other condition can one find the amount and abundance of blood in the spinal fluid.

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ABSTRACT OF DISCUSSION

DR. SANGER BROWN, Chicago: With reference to the first case reported, I should like to ask about the sense of taste in the anterior two-thirds of the tongue on the side of the facial anesthesia caused by involvement of the two upper branches of the fifth nerve. The diagnosis will, I think, have to be, as I presume is intended, a provisional one, that is, as to the actual cause giving rise to the symptoms. In view of the history I should not be surprised if, after all, we had to deal here with a case of brain tumor and cerebral anemia. The course of the symptoms would suggest the possibility of such a condition, and even though the diplopia

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perhaps and especially the vertigo and the very severe attack lasted only a few days, it is very likely that those symptoms were due to alteration in the blood-supply caused probably by the presence of a cerebral tumor. It seems most reasonable to me to assume that it is a tumor and that this is the cause of the persistence of the sensory disturbance in the region of the two upper branches of the fifth nerve. I think it is generally conceded now that the sense of taste in the anterior two-thirds of the tongue passes to the brain through Meckel's ganglion, which, of course, is connected with the second branch; therefore, one would hardly expect those symptoms. It would be inconceivable for me to think of a circulatory lesion, or any lesion except a small neoplasm, that would produce the persistence of so definite a focal symptom; whereas by assuming that there is a small neoplasm it would not be a long shot to suppose that its presence would cause just such a circulatory outbreak as seems certainly to have occurred in this case.

DR. CHARLES R. BALL, St. Paul, Minn.: I am somewhat surprised at the diagnosis of arteriosclerosis. It seems to me that the description gives an excellent picture of the symptoms of a vascular syphilitic endarteritis. The dizziness and occipital headache which preceded for some time the outbreak of the other symptoms; the glycosuria and polyuria, which are, as is now known, many times the accompaniments of vascular syphilitic endarteritis, particularly when the lesion is in the region of the inner peduncular space; the transient character of the paralysis and the great tendency to relapse, these relapses occurring over a period of a few months; the improvement, and the fact that by the multiple character of the lesion the mentality is so little impaired, would seem to me to make the diagnosis of arteriosclerosis very unusual. The really important observation in deciding the question definitely would have been an examination of the spinal fluid, which in all probability would have settled the diagnosis.

DR. H. E. CONLEY, Cannon Falls, Minn.: I followed Dr. Hammes carefully, wondering what he was going to do with the glycosuria, what relation this had to the trouble, whether a causative effect or simply a coincidence. It reminded me that some time ago I had a case in which some of these symptoms appeared, especially the recurring dizziness and the inclination to fall, with some headache but not so marked. It was some time before the diabetic condition was discovered, but as soon as it was, without other treatment than diet the dizziness entirely disappeared and the other symptoms were very much improved just by diet alone.

It would be interesting to have this question answered, if possible, in detail: What relation did the glycosuria bear to this trouble?

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DR. ROSS MOORE, Los Angeles: I should like to ask Dr. Hammes to tell us whether or not there was any respiratory symptomatology at the beginning of the attacks in this first case, which would be at all like that described by Dr. Kramer in his paper.

DR. C. EUGENE RIGGS: Four of these cases have been reported in the United States, two by Spiller in an article he presented on this subject before the American Neurological Society two years ago. The clinical syndrome of those two cases was exactly like that in the case presented this afternoon, and these two cases were followed by necropsy which proved conclusively that this was the type of trouble that we have to deal with. Of course, I am aware of the fact that only a necropsy would absolutely prove this contention. We were open to criticism with reference to not examining the spinal fluid. But sometimes, I think, the patient ought to be considered, and whenever there is an intracranial pressure or an intracranial neoplasm it is our practice never to make lumbar puncture.

DR. E. M. HAMMES: Answering Dr. Brown's question, the case was normal for the higher tongue, there being no response to taste. The possibility of brain tumor we considered very seriously, and excluded it on the history of four years' duration, on the absence of intracranial pressure, and on the fact that if the lesion, which undoubtedly lies in the cerebellar region or floor, were in the brain, the pressure would be great and we should get a choked disk in that time. Another point, which might exclude tumor is that since December the patient has not had any headache whatsoever.

Dr. Ball spoke of the multiple character of the lesion. It struck me that this lesion was interlocular as much as any lesion could be. The location must be such that it impinges on the glossopharyngeal nucleus. The pain and temperature sense caused by involvement of the proximal portion of the fifth nerve, and the transient paralysis of the sixth and seventh nerves is, I think, readily explained by the transitory circulatory disturbance and the close relation of the other nuclei. One reason why we did not perform lumbar puncture was the first result of diagnosis. We felt that the man had cerebral tumor with subsequent hemorrhage, and we thought that if we performed lumbar puncture at that time we might stir up trouble.

In regard to Dr. Moore's question, there was no respiratory disturbance when I saw the patient about twelve hours after the onset of the attack. The patient has given us the promise of a post-mortem when the time comes, so I hope that we shall be able to report more fully on the case in years to come.

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THE CONCEPTION OF HOMOSEXUALITY

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Of the abnormal sexual manifestations that one encounters none, perhaps, is so enigmatical and to the average person so abhorrent as homosexuality. I have discussed this subject with many broad-minded, intelligent professional men and laymen and have been surprised to hear how utterly disgusted they become at the very mention of the name and how little they understand the whole problem. Yet I must confess that only a few years ago I entertained similar feelings and opinions regarding this subject. I can well recall my first scientific encounter with the problem, ten years ago, when I met a homosexual who was a patient in the Central Islip State Hospital. Since then I have devoted a great deal of time to the study of this complicated phenomenon, and it is therefore no wonder that my ideas have undergone a marked change. *Tout comprendre c'est tout pardonner,* I have met and studied a large number of homosexuals and have been convinced that a great injustice is done to a large class of human beings, most of whom are far from being the degenerates they are commonly believed to be.

In his "Three Contributions to the Sexual Theory," Freud introduces two terms which are very useful in discussing sexual aberrations. He calls the person from whom the sexual attraction emanates the sexual object and the action toward which the impulse strives the sexual aim. Bearing in mind these terms, we may

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define homosexuality or uranism as that form of sexual aberration in which the sexual object is a person of one's own sex. That is, the sexual object of the homosexual man is not a woman but a man, and the sexual object of a homosexual woman is not a man but a woman. It is for that reason that such individuals are also referred to as contrary sexuals or inverts.

Most of the investigators agree that of the sexual aberrations homosexuality is by far the most wide-spread. It is very difficult, however, to give a correct estimate of the number of inverts. Many attempts have been made, notably by Magnus Hirschfeld,[1] who has had more experience with homosexuality than any other person. Hirschfeld estimates the number of male inverts of the population at about 1.5 per cent. Dr. v. Romer estimates that the city of Amsterdam contains about 1.9 per cent. inverts. Following the direction of Magnus Hirschfeld I attempted to find out the proportion of inverts in Greater New York. I invoked the aid of six cultured inverts who were strangers to one another, so that they moved in entirely different circles. They were very anxious to assist me in this work, but after about eight months' observation I found that the results differed to such an extent that it was impossible to compute any definite estimate. All that I can say is that there are many thousands of homosexuals in New York City among all classes of society. Homosexuality is not a product of big cities. When we read the works of I. Bloch, M. Hirschfeld, Moll, Havelock Ellis, and others, we are soon convinced that homosexuality is ubiquitous. One finds it among primitive and enlightened races during all epochs of history.

Nor is homosexuality confined to defectives, as is commonly supposed. Investigators agree that homosexuality is no sign of mental or physical degeneration.

1. Hirschfeld, Magnus: Das Ergebnis der statistischen Untersuchungen über den Prozentsatz der Homosexuellen, Jahrb. f. sexuelle Zwischenstufen, 1904, vi, 109.

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Thus Ivan Bloch† says: "I no longer entertain any doubt that homosexuality is compatible with perfect mental and physical health." This same author quotes Magnus Hirschfeld as saying that homosexuality may occur in persons just as healthy as normal heterosexual persons. Similar ideas are expressed by Näcke and others. My own findings concur with these views. Most of the inverts I know belong to our highest types both mentally and physically and show absolutely no hereditary taints. Without entering into a detailed discussion of this question I will say that I am convinced that homosexuality as such is entirely independent of any defective heredity or other degenerative trends.

Inverts have been variously classified by different investigators of the subject, notably by Kiernan, Lydston, Krafft-Ebing, Hirschfeld, Bloch and others, but for our purpose it will suffice to mention that there are three classes:

1. Absolute inverts whose sexual object must always be of the same sex. Most of them entertain a *horror feminae* or are impotent when it comes to the performance of the normal heterosexual act. I saw a number of patients, who were ignorant of their inversion, who first consulted a physician for psychosexual impotence.

2. Amphigenous inverts (psychosexual hermaphrodites) in whom the inversion lacks the character of exclusiveness, and hence their sexual object may belong to either sex.

3. Occasional inverts who resort to homosexuality under certain external conditions, especially in case the normal sexual object is inaccessible. Such individuals are able to obtain sexual gratification from a person of the same sex.[2]

It is interesting to note how the inverts themselves view their inversion. Some take it as a matter of course and demand the same rights as the normal. They are

† Bloch, Ivan: Das Sexualleben unserer Zeit, Marcus, Berlin, 1909, p. 543.

2. Freud: Three Contributions to Sexual Theory, p. 2.

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perfectly contented with their lot, and seldom consult a physician. "I would not for the world have anybody interfere with my personality; I just wish to consult you about a *modus vivendi* for myself," writes a young inverted clergyman on asking me for an appointment. Some, however, struggle against it and consider it a morbid manifestation. It is only the latter who can be helped by treatment.

There may be some congenital inverts, but of the forty-nine cases that I have analyzed I always discovered one or more early affective sexual impressions which favored the development of homosexuality. In others a fixation of the inversion took place earlier or later in life through external favoring and inhibitory influences, such as exclusive relations with the same sex in boarding-schools, in the army, in the navy, in prison, etc. It is no simple matter to find these early unconscious impressions. It usually takes weeks and months of psychanalysis before they can be discovered. It is therefore comprehensible why such cases have been called congenital. It is only of late that some of these cases have been studied psychanalytically by Freud, Sadger and a few others.[3] Another point against the assumption of congenitality is the fact that hypnotism and other psychotherapeutic means may cure the inversion, which is hardly possible if it were congenital. Indeed, when we examine the literature on this particular point, we find that the authors are far from being in accord on the question of whether inversions are congenital or acquired. At first it was supposed that homosexuality was simply a vice acquired through excesses or through a suggestion in early life (Binet, Schrenck-Notzing). Krafft-Ebing assumed a congenital and an acquired form. Since then there has been a tendency to limit the acquired form as evidenced in the works of Moll and others. Hirschfeld assumes that homosexual-

3. Similar views are expressed by Coriat in his paper on Homosexuality, New York Med. Jour., March, 1913.

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ity always contains a congenital element. Näcke refuses to recognize a congenital and acquired homosexuality but suggests instead the true and false inversions. He also describes that form which manifests itself late in life as tardive homosexuality and maintains that it is not acquired but based on a congenital basis. All these diversities are, in my opinion, due to the fact that none of these authors have gone deep enough with their patients.

When we examine the theories advanced concerning the nature of inversion, we are soon confronted with the theory of hermaphroditism, which was brought into prominence by Lydston, Kiernan and Chevalier. It starts with the fact of anatomic hermaphroditism and shows that a certain degree of it really belongs to the normal. This leads to the conception of the original predisposition to bisexuality which changes in the course of development to monosexuality, leaving slight remnants of the stunted sex. This conception was then transferred to the psychic spheres and the inversion was conceived as an expression of psychic hermaphroditism. But to confirm this it would be necessary to find a regular correspondence between the inversion and the psychic and somatic signs of hermaphrodism which was not realized. Although one frequently finds in inverts a diminution of the sexual impulse and a slight anatomic stunting of the organs, it is by no means a regular or preponderate occurrence, so that one is forced to conclude that there is no relation between homosexuality and somatic hermaphrodism.

Many observers lay a great deal of stress on the so-called secondary and tertiary sex characteristics which are supposed to occur in inverts. Thus Hirschfeld, who bases his experience on 1,500 inverts, asserts that he never saw a homosexual who did not differ from a perfect man. As much as I respect the opinion of Dr. Hirschfeld I must say that I cannot quite agree with him. In my little experience I have seen many homo-

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sexuals without any of the secondary sex characters. I will admit, however, that I have not examined my patients as carefully as Hirschfeld has his. On the other hand I know that some men show some of the secondary sex characters who are absolutely heterosexual.

The bisexual theory expounded by Ulrich—*anima muliebris in corpore virili inclusa*—is entirely untenable. The same may be said of Krafft-Ebing's theory that the bisexual predisposition gives to the individual male and female brain-cells somatic sexual organs which develop toward puberty under the influence of the independent sex glands. All that can be said is that, although a bisexual predisposition may also be presumed for the inversion, we do not know wherein it exists beyond the anatomic formations and that we are dealing with disturbances experienced by the sexual impulse during its development.[4]

According to the theory of psychic hermaphroditism the sexual object of inverts would be the reverse of the normal. Thus the invert would succumb to the charms emanating from the manly qualities of body and mind; he would feel like a woman and look for the man. This conception, although true in a great many cases, does not by any means indicate the general character of the inversion. Many homosexuals retain their virility and look for feminine psychic features in their sexual object. Freud demonstrates this nicely by mentioning the fact that masculine prostitutes in offering themselves to inverts imitate to-day, as in antiquity, the dress and the attitudes of the woman. Moreover, among the Greeks, who numbered among their inverts some of the most manly men, it was surely not the masculine traits of the boy that attracted them but rather his physical resemblance to the woman as well as his feminine psychic qualities, such as shyness and demureness. When the boy grew up he ceased to be a sexual object for men

4. Freud: Three Contributions to the Sexual Theory, p. 9.

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and in turn became a lover of boys. All this goes to show that the sexual object in this case, as in many others, is not of the same sex, but that it unites both sex characters. It is a compromise between the impulses striving for the man and for the woman, but firmly conditioned by the masculinity of the body (the genitals). I purposely paraphrased Freud, as these points will have to be borne in mind later.

The sexual aim of inverts shows no uniformity. The popular idea of homosexual relations presupposes that inverts always practice fellatio or intercourse per anum. As a matter of fact these sexual aims are least practiced. Many homosexuals are as disgusted at the mention of these practices as normals. Some content themselves with an effusion of feelings. Ten of my analyzed cases never had any sexual relations with their sexual object. Some practiced mutual masturbation, others coitus *inter femora.*

Strange as it may seem, the diagnosis of homosexuality is not always an easy matter. In the first place it must be urged that a sporadic homosexual act does not necessarily mean homosexuality, nor does the absence of such acts signify heterosexuality. There is naturally no difficulty when one is confronted with an absolute invert who acknowledges his inversion. There are, however, a number of inverts who are really ignorant of their inversion. Eleven out of my forty-nine patients did not realize that they were homosexual, although nearly all of them had had homosexual experiences some time in their lives. They sought treatment for psychosexual impotence or for some neurosis. I have also seen patients who were treated for a long time for psychosexual impotence by prostatic massage, etc., who were all the time aware of their inversion. They kept silent because the treatment gave them pleasure or because they were ashamed or afraid to tell the doctor the true state of affairs. For many reasons the average doctor is not especially affable to a homosexual patient, and many

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a sensitive invert has had cause to regret his confidence in the doctor.

The diagnosis should be based on the somatic and psychic elements of the case, especially the latter. Naturally the psychanalyst finds it easier to diagnose a difficult case than one who does not enter into the deeper psychologic mechanisms. Dreams are usually an excellent guide in the diagnosis of homosexuality, but it must be remembered that they should be judged by the latent and not by the manifest content by which Näcke judges them. I have analyzed many apparently sexual dreams whose latent content showed a homosexual wish.[5] We must also remember that not all erotic dreams of homosexuals are homosexual,[6] and that some apparently homosexual dreams have nothing to do with homosexuality as an inversion.[7] Furthermore, many homosexuals who are anxious to become heterosexual often show normal dreams; the dreams simply realize their wishes. I have observed this mechanism in many homosexuals, and it is for that reason that I cannot agree with Dr. Coriat, who states that the "dreams furnish us not only the best, but the *most incontrovertible,* [the italics are mine] evidence of the result of treatment." There is a class of patients who do not show the characteristics of the invert who are nevertheless constantly afraid of becoming homosexual or fear lest some one should suspect them of homosexuality. I have seen a number of such patients who were classed as homosexuals. I also saw one of these patients in consultation with Dr. Hirschfeld, who diagnosed the case as a severe psychasthenia and saw nothing homosexual in the case. If such patients are questioned, one will find that they never were in love with any person of the same sex —an important diagnostic point—and they show besides many symptoms that one does not find in the inver-

5. Brill: Psychanalysis; Its Theories and Practical Application, p. 55.

6. Moll: Handbuch der Sexualwissenschaften, p. 654.

7. Freud: Ueber Infantile Sexualtheorien Sammlung Kleiner Schriften zur Neurosenlehre, Zweite Folge, Deuticke, Wien.

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sions. These patients may be called unconscious homosexuals, and they often develop into paranoid states, a discussion of which cannot here be entered into.[8] I may add that the patient seen with Dr. Hirschfeld is now suffering from the paranoid form of dementia praecox.

As we are dealing with a psychic manifestation, the hope for a cure of homosexuality lies in psychotherapy. I can never comprehend why physicians invariably resort to bladder washing and rectal massage when they are consulted by homosexuals, unless it be to kill the homosexual cells in the prostate so that their place may be taken by heterosexual cells, as one physician expressed himself when one of my patients asked him how massage of the prostate would cure his inversion. It is an unfortunate fact that such ridiculous ideas are often heard in the discussion of psychosexual disturbances. Only a few months ago a patient told me that he was told by two physicians that his hope for a cure lay in castration.

When hypnosis came into vogue a great many workers in this field utilized it in the treatment of homosexuality.[9] It was soon found that it failed to come up to expectation. Some patients could not be hypnotized, others suffered relapses, and still others did not react to the suggestions. Indeed, very few sexologists place much trust in hypnotism as a cure for inversions. Of late Moll has advanced a new psychotherapeutic method which he calls the association therapy. It consists of a methodical development of the normal and a methodical suppression of the perverse associations.[10] It is too early to speak of the merits of this treatment; so far as my knowledge goes no one has used it besides Moll. In the treatment of my cases I use exclusively psychanalysis. Freud, Sadger and others, have used this method

8. Brill: Psychanalysis; Its Theories and Practical Application, chap. VI.
9. Compare the works of Krafft-Ebing and Schrenck-Notzing.
10. See Moll, Handbuch der Sexualwissenschaften, p. 662.

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for a number of years,[11] and the results obtained are very gratifying. Besides, psychanalysis has the advantage over the other psychotherapeutic means in so far as it enters into the deeper mechanisms of the phenomena, and, although we have not yet a full explanation of the origin of inversions, it has revealed the psychic mechanism of its genesis and has essentially enriched the problem.

Those who are acquainted with the principles of psychanalysis will recall the close connection between the neuroses and the perversions. Every neurosis regularly shows some admixture of inversion, and during the analysis of a hysteria or compulsion neurosis one invariably finds some heterosexual and some homosexual roots. When we analyze a case of inversion we find that the masculine ideals of the invert regularly conceal the early infantile feminine ideals, usually the mother or foster-mother, which succumbed to repression at a very early age. Before the age of puberty the sexual feelings are usually unspecialized and the persistent desire for the man usually makes its appearance at or shortly before puberty. The lasting homosexual desire is usually brought about by the fact that the mother loses her rôle as an ideal forever, or for a long time, either through death, illness, or other estrangement. The later homosexual may then turn to his father, older brother or some other older man of his environment. Among the homosexual ideals, and besides the homosexual and heterosexual features hitherto desired, one's own person, one's own image, plays a great part; in other words, the road to homosexuality always passes over narcissism, that is, love for one's self. The stage of narcissism is characterized by the fact that the developing individual, while collecting into a unit his active

11. Freud: Three Contributions to the Sexual Theory, Eine Kindheitserrinrerung des Leonardo da Vinci. Sadger: Fragment der Psychoanalyse eines Homosexuellen, Jahrbuch f. sexuelle Zwischenstufen, ix; Ein Fall von multipler Perversion mit hysterischen Abszenen, Jahrb. f. psychoanal. and psychopath. Forschungen, ii; and Ist die Konträre Sexual Empfindung heilbar? Ztschr. f. Sexualwissensch., 1908.

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auto-erotic sexual impulses in order to gain the love object, takes first himself, his own body, as the object, before going over to the object selection of a strange person. Narcissism is therefore a necessary stage of development in the transition from auto-erotism to the later love object. The love for one's own person, which only conceals the love for one's own genitals, represents a stage of development which is always present, and in a great many persons lasts a long time. The remaining road later leads to the choice of objects with similar genitals. As Sadger puts it:

> **Every man usually has two primary and primitive sexual objects, and his future life depends on whether or not he finally remains fixed, and on which of the two the fixation takes place. For the man these two objects are his mother or foster-mother and his own person. To remain healthy he must rid himself of both, and not tarry too long with either of them.[12]**

It is assumed that the invert could not get away from himself, that is, he was unable to free himself from the desire of requiring genitals similar to his own in the love object. He is more successful, however, in freeing himself from his mother-image which is brought about by identifying himself with her and thus taking himself as the sexual object. With the repression of the love for the mother there occurs a repression of love for all womankind. According to Sadger it follows the following trend of thought: "If the best of all women, my own mother, amounts to so little, how could any other woman stand the test?"[12] As soon as the analysis is entered on, one often finds that inverts are not at all indifferent to the charms of women, but as soon as any excitation is evoked by the woman it is at once transferred to a male object. This mechanism which gave origin to the inversion is thus repeated throughout life and their obsessive striving for the man proves to be determined by their restless flight from the woman.

12. Sadger: Ein Fall von multipler Perversion, p. 112.

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It is also noteworthy that most inverts are only or favorite children. I have shown elsewhere that such children are usually overburdened with love[13] and hence remain insatiable for the rest of their lives. This accounts for the fact that when they tear themselves away from their mothers they often reject the whole sex.

These are some of the salient points brought out through the analysis of inverts which I shall illustrate by the following cases:

CASE 1.—W., aged 40, single, American, was referred to me for treatment by Dr. Frederick Peterson in December, 1910. He was an absolute invert, having attempted sexual intercourse once at the suggestion of his valet and failed. He showed some of the secondary sex characters. He had a very delicate skin, of which he was proud because it was just like his mother's, a scanty growth of hair on his face, and narrow shoulders, and broad hips. Psychically he recalled an old maid. He was very neurasthenic and crabbed, but his mood often changed to a feeling of self-sacrifice and marked consideration for others. He was very artistic, loved music, pictures, and took a great interest in architecture. He had had many homosexual experiences; he was loved by and loved men, and never entertained any sexual feeling for women. As soon as I entered into his life I found that he had a striking polymorphous perverse sexuality which continued into the age of puberty. His desire for looking was especially strong. At the age of from 6 to 8 he used to lock himself in the bathroom and look at himself naked in the mirror. He often put the mirror on the floor and excited himself by looking at his penis. Although he at first recalled no heterosexual experience, he later recalled many such incidents. Thus at the age of 6, while visiting a relative, he slept with a servant who practiced masturbation with him. She also taught him sexual intercourse. Five years later at the age of 11 years he met this servant and attempted intercourse with her. At the same age he had a number of sexual experiences with a girl of 14 years. His homosexual experiences began at the age of 9 years when he was taught fellatio by a classmate. He was his mother's pet. When he was young he was very much attached to her, but after the age of puberty he could never be with her without quarreling. His father he openly hated. I should like to give you a full analysis of this very interesting case, but I shall reserve this for another occa-

13. Brill: The Only or Favorite Child in Adult Life, New York State Jour. Med., September, 1912; reprinted as Chapter X in Psychanalysis, Its Theories and Practical Application, Saunders Publishing Co., Philadelphia.

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sion, and will simply say that this case demonstrates with absolute certainty the psychologic mechanisms found by Freud and Sadger. After six months' treatment the patient left me perfectly cured and has remained so ever since.

CASE 2.—O., 46 years old, single, American, was referred to me for treatment for psychosexual impotence by Dr. W. S. Reynolds in the beginning of May, 1909. The patient stated that he attempted intercourse at the age of 22 years and failed, and since then had been unable to get an erection without being helped by friction. For about a year before coming to me he attempted intercourse three times, for experimental reasons as he put it, and succeeded in getting only "half an erection." The patient was somewhat shy, and of the plethoric type. He gave a clear account of his life and soon became interested in the analysis. After studying him for two weeks I discovered that his impotence was due to homosexuality. His *vita sexualis* was characterized by a rather prolonged infantile sexuality. He wet the bed up to the age of 13 years. Between the ages of 6 and 8 years he practiced exhibitionism with a little girl. At the age of 12 years he began to masturbate, a practice which he continued to the time of treatment with Dr. Reynolds. He attempted heterosexual intercourse at the age of 22 years, and his first homosexual experience began at the age of 29 years and continued on and off whenever the occasion presented itself. These experiences were always accompanied by conflicts and feelings of remorse. When young he was very fond of his mother, but since the age of 8 years she had disgusted him because "she gave birth to so many children." This patient showed no secondary sex characters, although he himself thought that he did not have enough hair on his face and that his penis was small. His penis was slightly below the average. When I first discovered the patient's homosexuality I took a rather gloomy view of the prognosis. My reason for feeling so was that he entertained some vague ideas of reference. He imagined that whenever he came near men they made certain motions which meant to him that they considered him effeminate, but after ten months' treatment the patient left me cured. The analysis demonstrated almost all the homosexual mechanisms.

CASE 3.—Patient, aged 28 years, single, American, actor, was referred to me by Dr. Frederick Peterson in the beginning of March, 1912. The patient was anxious to be cured of homosexuality. He himself thought that it was congenital, as he recalled that he became sexually excited at the age of 5 years on the occasion of sleeping with his father. He showed no secondary sex characters, and analysis showed that for some time before this episode there was an affective sexual experience which prepared the soil for the later development of the inversion. This case was especially interesting because besides the homosexuality the patient also evinced very strong

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sadistic perversions. He entertained many sadistic fancies. He longed for the days when the people went around armed with dirks and daggers, and he usually carried a revolver in his pocket. He spent considerable time in the menagerie in front of the tiger's cage. He loved to see the tiger excited and to hear him roar and he imagined that he exerted a certain influence over the tiger. The analysis brought out all the mechanisms found by Freud and Sadger in their inverts, and after six months treatment the patient was discharged as cured, and has remained perfectly normal since.

I could cite a number of other cases of the different forms of homosexuality which I have cured by psychanalysis, but, as it is impossible to give here more than a few lines of cases that would consume hours of reading, I will close my paper with the wish that it may serve to throw some light on these obscure phenomena and remove some of the prejudices to which these unfortunates are subjected.

ABSTRACT OF DISCUSSION

DR. D'ORSAY HECHT, Chicago: I should like to ask Dr. Brill whether or not he made any special inquiry into the heredity of these homosexuals. As I recall it, he stated that they did not show any particular inheritance that could be of a degenerative type. I do not ask because I question his statement, but because, after all, I think that there is always a latitude in the interpretation of what constitutes inheritance, as to whether or not a person could inherit a certain tendency not rationally acquired, but having a physical basis which would permit these sexual perversions to take root and remain for all time.

I was also impressed with the effort of Dr. Brill to correct homosexuality by decrying it. But if in the eye of the specialist homosexuality is but a contravention, socially speaking, and if it has just as much right to a hearing from the point of view of a sexual act as has heterosexuality, I really cannot see why the homosexual should care to be delivered from his homosexuality, except that he feels disgraced by it. Then again, a large number of homosexuals are in no way abhorrent of themselves in respect to their natures; they seem to be perfectly happy and perfectly well adjusted, probably in a restricted sense, and these patients probably are not worth while treating as Dr. Brill treats them. If we accept homosexuality as a condition which has as much right to exist as heterosexuality, why should we address ourselves to the duty of treating it?

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I am wondering, too, whether or not the antisocial attitude toward the homosexual individual is, so far as the law is concerned, out of all proportion to the offense as it is conceived by the medical man. It is a fact that in Great Britain, for instance, the attitude on the part of the British law toward the offense of sodomy as a social crime is tremendously severe. But there must be something in homosexuality much more potential than merely the fact that the homosexuals are attracted by certain special attributes of the male, as stated by Dr. Brill; there must be something in homosexuality that far transcends what Dr. Brill conceives as homosexuality. I would ask Dr. Brill if the law takes this into consideration when it comes to the application of punishment for these offenses against society, such as, for instance, we have seen so well characterized in the celebrated case against Oscar Wilde. That, of course, was one of the most flagrant offenses of the kind, at least in the stratum of society in which it was practiced, of which we have knowledge.

DR. HOWELL T. PERSHING, Denver: I should like to ask Dr. Brill, when he has made the diagnosis and satisfied himself of the origin of the perversion, in what way he treats it.

DR. ROSS MOORE, Los Angeles: One point that occurred to me as Dr. Brill was reading, and which was not particularly clear to me, was this: In the early part of his paper he spoke about a number of patients coming to him for some psychosis or for impotence, who did not know before they came to him that they were homosexual, and I wondered whether Dr. Brill, or any of us in carrying on such work as Dr. Brill has carried on in his cases, might not be in danger of doing society an injury as society sees the matter, or the patient an injury, by suggesting to him the possibility of homosexual conditions —whether or not it is a cure to make the man understand that he is homosexual.

I want to ask also how much of the real fundamental psychanalysis according to Croy he used in these cases. I have wondered whether or not he has modified the idea of psychanalysis, as ordinarily practiced, etc., in the handling of these cases. I should be very glad to have him tell us a little about it.

DR. ALBERT E. STERNE, Indianapolis: I have found it difficult to reconcile the statement Dr. Brill has made, that these individuals were not mentally or physically in any sense alienated from that which is commonly regarded as normal, with the impression which I gained that there is a distinct abnormality presented by the individuals in the three cases reported in detail. It is very hard to reconcile oneself to experiences in conditions of this kind and not recognize certain collateral conditions that have been manifested by these indi-

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viduals; in other words, to look on this homosexuality as an isolated phenomenon. We are not accustomed, of course, to regard this abnormality as a manifestation of a mental condition, while we do regard it as a marked evidence of degeneracy. This subject has, of course, considerable ethical importance.

It is hard for me again to realize, not only from the paper that Dr. Brill has given us, but also in personal conversations with him, how he can feel satisfied, and how he can convince others, that these individuals are absolutely cured. He simply takes the statement of the patient that he no longer indulges in the practice that he had earlier acknowledged. That may be convincing to Dr. Brill, but it is pretty difficult at a distance to be convinced.

DR. A. A. BRILL, New York: In some cases of homosexuality I found no hereditary factors; in other cases there were some. Most of the homosexuals whom I have seen did not seek treatment and were perfectly content to remain as they were. I have also seen some who wished to be heterosexual, but, as they showed degenerative trends, I did not care to undertake the treatment. Altogether, there are comparatively few homosexuals who seek treatment and who can be made heterosexual. I never undertake treatment unless the patient is mentally normal, and I always take care to explain to him that it will take a long time, say from six months to one year, to bring about a recovery. Unless all such conditions can be fulfilled it is useless to undertake the work.

The only way that I could answer Dr. Pershing's question would be to give him a detailed analysis of a typical case, but this naturally is out of the question here. The analysis does not differ technically from any other psychanalysis, but special attention must be paid to the homosexual mechanisms.

Answering Dr. Moore's question, I would say that it is impossible to suggest to any one that he is homosexual when he is not. The patient may not know the meaning of the term, and a great many do not know that there is such a class as homosexuals, but they are always aware that they have sexual feelings for persons of the same sex. A patient was once sent to me by a physician because he suffered from compulsions. Nothing was said about homosexuality. After a few sessions I became convinced that the patient's neurosis was based on homosexual conflicts. I told him that I thought he was homosexual. He was surprised to hear the word, said that he did not know its meaning and I was obliged to explain what homosexuality meant. He soon confirmed my diagnosis by relating to me many homosexual experiences, such as wrestling with specially selected persons while at college which usually caused ejaculations. He also told me of numerous compulsive ideas of a homosexual nature which he had

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never mentioned to his former physicians. There was no question about his homosexuality. His whole history was quite typical of the condition. Of course, one must be particularly careful not to suggest anything to the patient and, as a matter of fact, we psychanalysts are especially careful to exclude all suggestions; on the other hand, when I am sure that the person is homosexual I do not hesitate to tell him so.

Answering Dr. Sterne's question as to how I can determine when a patient is cured, I would say that when a patient comes to me and gives a history of having been actively homosexual almost all his life and describes the terrible conflicts he has gone through and then, in the course of treatment, gradually begins to take interest in women and eventually reacts heterosexually just as he formerly did homosexually, I have no reason to doubt the man's statements or his recovery. It should not be forgotten, moreover, that he did not come to me at my solicitation, that he was anxious to be cured, that I followed his case for months, or perhaps for more than a year, that I know him better than any one else in the world and that there is no reason why he should lie about his condition.

I did not say that all homosexuals are normal mentally and physically, but I do maintain that they do not differ in that respect from heterosexuals and, in my experience, they have belonged to the finest types.

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INTRACRANIAL DIVISION OF THE AUDITORY NERVE FOR PERSISTENT TINNITUS

CHARLES H. FRAZIER, M.D.
PHILADELPHIA

The surgery of the cranial nerves offers an interesting field for those who have made the treatment of nervous lesions an object of especial study. Perhaps because the symptoms to which these lesions give rise are not of serious moment in that they do not threaten life, or whatever may be the explanation, the fact remains that an insignificant number of people avail themselves of the relief that surgery affords. How many there are, who for a greater part of their lives endure the unpleasant disfigurement that follows a complete facial palsy, without so much as considering the possibilities of correction that surgery offers! And yet the facio-hypoglossal or the facio-accessory anastomosis is an operation carefully planned and fully justified by the results. Similarly with trigeminal neuralgia, it may be said that whether because of ignorance or indifference on the part of the profession or prejudice on the part of the patient, there are a surprising number who for years suffer as excruciatingly as they do needlessly. It is not an exaggeration to say that the majority of patients with trigeminal neuralgia sent to me for advice and treatment have been victims of the disease for at least ten years and many for twice as many, and this in face of the fact that absolute relief may in many instances be afforded without resort to a general anesthetic or the risks, real or imaginary, of an operative procedure, as is the case with alcoholic injections.

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I have taken this opportunity to dwell on the delay and procrastination in resorting to surgical procedure, because it seems to me a matter of very considerable moment. There is a woeful indifference to the actual needs and comfort of patients, not only those suffering from conditions already mentioned, but from tumors and lesions associated with increased intracranial tension. The records of cases in my clinic reveal the rather pathetic situation of two out of three patients already blind, that is with an optic atrophy so far advanced that recovery of vision is entirely out of the question. Of all the benefits to be derived from operative procedures in this class of cases, preservation of vision is perhaps the greatest and at the same time the one most longed for by the patient. Now, the surgeon does not see these cases, as a rule, as soon as the neurologist, and the latter has the greater opportunity of impressing on the family physician the obligation, clearly his, to lay clearly and fairly before the patient the possible accomplishments of surgical therapy. The so-called expectant form of treatment so popular and so delusive is, as Sir Victor Horsley says, not treatment at all, but in most instances a tacit acknowledgment on the part of the physician that he has been unable to make a diagnosis or, if not, to inaugurate an effective course of treatment.

Up to the present time surgical therapy has been applicable to the treatment of functional disturbances in only three of the cranial nerves—the trigeminal, facial and auditory—and of these the auditory nerve has been the last to be brought within the scope of surgical procedure. Now what has been said of the infrequent resort to surgical measures for the relief of lesions of the trigeminal and facial nerves may be said with equal, if not greater, force, of the eighth or auditory nerve.

The operation which I am about to describe is the intracranial division of the auditory nerve. There are

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several circumstances in which this procedure may be resorted to with propriety. In 1908, Dr. Charles K. Mills discussed the propriety of this operation for the relief of aural vertigo, and so far as I know the operation had not been thought of before in this connection. His patient was a woman in her fifty-fifth year, and the method of procedure and the outcome of the operation have already been made a matter of record.[1] One naturally inquires whether as much relief might not be derived from the removal of the semicircular canals, an operation perhaps less formidable, and therefore to be given preference. There is no doubt but that the latter operation should be selected when the vertigo is unassociated with tinnitus, but it so happens that in many instances we find the vertigo and tinnitus combined, and it is for this particular situation that the operation on the auditory nerve is more specifically indicated.

Consideration should be given to relief that might be afforded certain forms of persistent otalgia by division of the auditory nerve. So far as I know there are no available records in which the effect of this operation has been tried, and the situation is somewhat complicated by the fact that, as Hunt[2] has pointed out, there may be, as one of the underlying factors, an associated lesion of the nervus facialis. We are all familiar with the clinical syndrome which Hunt has elaborated from his careful and original studies of the sensory mechanism of this nerve through its various connections; the relation of the geniculate ganglion to the interior of the auricle and the external auditory canal, the connection of the ganglion with the tympanic plexus, etc. If, as Hunt maintains, there may be in certain cases of persistent otalgia a neuritis of both the seventh and eighth nerves, it would be necessary to divide them both. To be called on to divide both nerves would simplify rather than complicate the intracranial

1. Surg., Gynec. and Obst., November, 1912.
2. Jour. Nerv. and Ment. Dis., February, 1907, and June, 1909.

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technic, and the inevitable facial palsy might be considered an unsurmountable objection, although the patient might prefer the consequent facial asymmetry to the continuation of his affliction. In this connection, however, it is worthy of mention that in some instances a preexisting facial palsy is a part of the syndrome, and under such circumstances the facial nerve could be divided with impunity.

For final consideration of the indications for this operation, there remain cases of persistent and intractable tinnitus. Ordinarily, tinnitus aurium responds to local treatment, but the cases to which I have reference are those of a type so severe as to be the underlying cause of grave neurasthenia or serious mental disturbances, and both have been the cause of threatened suicide. In some instances, the tinnitus may be the only affliction; in others it is associated with vertigo or otalgia. To be sure, the selection of cases is a matter of considerable importance, and whenever possible those of central origin should be excluded. The appropriate cases are those in which the disease is labyrinthine in origin; it may originate in the vestibular ganglion, in the cochlear ganglion or in both, but as there is no means of separating the cochlear from the vestibular division, the entire auditory trunk must be sacrificed. It may be said further that the most promising cases are those in which there is loss of air conduction, with preservation of bone conduction, cases with definite cochlear lesions in which the tinnitus is low-pitched.

No doubt the deafness resulting from complete section of the auditory nerve has occurred to you as a possible objection to the operation, and this would have to be given consideration were it not for the fact that as a rule the patient is already deaf on the affected side.

Time will not permit of a consideration of the conditions which are responsible for the development of tinnitus, but I should like here to refer to an interesting case which, within the last year, was brought to my attention:

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History.—The patient, a comparatively young man, in his thirty-fourth year, was sent to my clinic by Dr. L. C. Suttner of Walla Walla, Washington. He had been struck on the head by a falling limb of a tree. Immediately after the accident he became unconscious and remained in this state for five hours. Almost immediately after regaining consciousness, five hours later, he observed a ringing in his right ear. On leaving his bed he noticed that in going up or down stairs, when standing with eyes closed, or in turning his head suddenly, particularly to the right side, he became dizzy and occasionally became nauseated. In addition, there was impairment of sensation in the distribution of the trigeminal nerve, particularly in the third division, and complete loss of vision on the affected side.

Physical Examination.—Patient is well-developed, robust and well-nourished. Skin is normal; no cyanosis, jaundice, edema or eruptions. Arteries are not sclerosed. There is a sense of numbness over the left temporal region, extending down below the molar eminence and even to the left side of the nose. Patient can close his eyes, wrinkle his forehead, contract his masseters, show his teeth, pucker the corners of his mouth, equally and well on both sides.

Heart: Outline normal; sounds regular and of good muscular quality; suggestion of systolic murmer at the apex. Abdomen negative.

Extremities (lower): Good muscular force; no incoordination; knee-jerks prompt; no ankle clonus; no Babinski. Gait seems somewhat uncertain. Station negative. Extremities (upper): Normal; no incoordination.

Blood: Red blood-cells, 5,500,000; white blood-cells, 13,200; hemoglobin, 87; polymorphonuclears, 65; lymphocytes, 31; large mononuclear, 1; transitionals, 2; eosinophil, 1.

Urine: Amber; light flocculent sediment specific gravity 1.025; slight acid; no albumin or sugar; no casts, cylindroids, or red corpuscles; considerable mucus; a few leukocytes; squamous epithelium; calcium oxalate crystals.

Examination of the ears by Dr. Randall: There is some nerve deafness in the left ear, namely, for low tones, as he claims to hear the 2,000 d. v. s. fork and many tones (notes) of the Galton whistle. His left naris was stuffy, a possible source of his tinnitus, but its freeing gave no relief, nor did the Politzer inflation or massage. His left drumhead is scarred, but with little trouble remaining. There is no objective bruit as of aneurysm. His injury would seem to have been on this side and to have pinched the fifth and eighth nerves.

Examination of eyes by Dr. de Schweinitz: Right eye: normal; vision 6/5. Left eye: optic nerve atrophy; probably

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secondary to lesion at base. Pupils react. No muscle palsies. Ocular symptoms suggests basal lesion anterior to chiasm.

Blood-Pressure: Systolic 125; diastolic 80.

Diagnosis.—Unquestionably, there must have been a fracture of the base, beginning in the petrous bone and extending forward to the optic foramen; for in no other way could one account for the trauma to the optic, trigeminal, and auditory nerves. The associated injury of these three nerves is a very unusual one, and in my experience quite unique. It is not common to find a simultaneous injury to the nerves of the posterior fossa and one of the anterior fossa, and while this is worthy of comment in passing, it is still more uncommon, at least in my experience, to see a persistent lesion of the trigeminal nerve following cranial trauma, and, furthermore, to see the facial nerve escape altogether when the injury has been extensive enough to involve both the trigeminal and auditory.

It was for the relief of the tinnitus, which had become quite unbearable and seriously impaired the patient's health, that I was consulted, and inasmuch as there was every reason to believe that the tinnitus was not central in origin, and as the hearing was already impaired on the affected side, I had no hesitancy in recommending to the patient section of the auditory trunk. To this he readily consented, and I proceeded somewhat as follows.

Operation.—A flap was reflected so as to uncover the occipital bone to the left of the median line and below the superior curved line. That portion of the occipital bone, between the emissary sinus and the median line, and from the level of the lateral sinus downward for 3 cm., was removed with rongeur forceps. On the reflection of a dural flap, a brain retractor was introduced beneath the cerebellar hemisphere, which was gently elevated to allow of the escape of cerebrospinal fluid from the cisterna magna. At the same time I instructed my assistant to puncture the dural sac in the lumbar region, but the withdrawal of cerebrospinal fluid in this way did not allow any greater displacement of the hemisphere, which was considered possible. The latter was gently displaced backward and gradually the petrous bone was uncovered as far as the internal auditory meatus. (See Fig. 1.) With good artificial illumination, the auditory and facial nerves were identified. The auditory was readily separated from the facial nerve and divided with alligator scissors. An electrode applied to the facial nerve gave an immediate response. The dural wound was closed with interrupted silk sutures and the musculocutaneous flap restored. At no time during the operation was hemorrhage a troublesome feature; the blood-pressure varied from 110 to 140 Hg. and the pulse from 80 to 105. The patient's condition at the conclusion of the operation

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was entirely satisfactory, and his convalescence, though prolonged by an incidental attack of appendicitis and tonsillitis, was otherwise uneventful. (See Fig. 2.) The effects of the operation were more than gratifying to the patient, as the intense roaring sound had disappeared.

CAUTION TO OPERATORS

While I have already indicated quite specifically the conditions under which this operation may be performed with propriety, one restriction should be insisted on, namely, that the operation be entrusted only to the hands of those who are familiar with problems of the surgery of the posterior fossa. I say this with some hesitation and with due regard for the accomplishments of surgeons skilled in other fields, but I am convinced that to be able to meet the difficulties peculiar to procedures in the posterior fossa effectively and with due regard for the patient's safety, one must have made them an object of special study and have the confidence that comes only with experience. The administration of the anesthetic by intratracheal insufflation offers many advantages. The appliances for securing the proper posture of the patient are absolutely essential; the control of hemorrhage requires one to be especially resourceful; and the subsequent manipulation of the cerebellum demands nicety of judgment. The displacement of the cerebellum must be effected without undue trauma on the adjacent pons and medulla and should not be attempted without taking advantage of the additional space to be derived by evacuation of the large basal cisternae. The separation of the auditory from the facial nerve requires patience, the use of artificial illumination is almost indispensable, and the assistance to be derived from electrical stimulation should always be availed of before the auditory nerve is divided. Although the auditory nerve is larger than the facial, no chances should be taken until the facial nerve is identified by electrical stimulation. I approach these operations not without due regard for the intrica-

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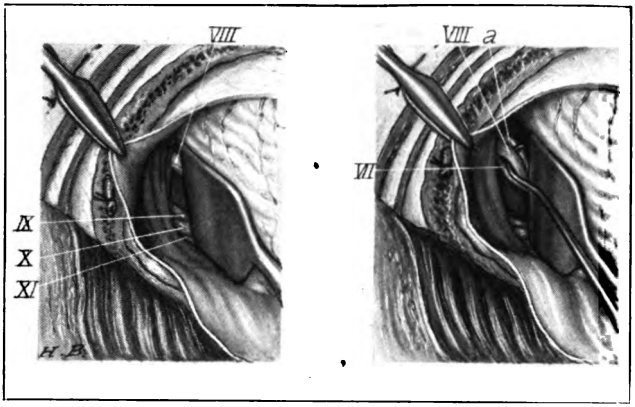

Fig. 1.—Left figure: Illustration showing the retraction of the cerebellar hemisphere and the relation of the auditory nerve at the internal auditory meatus to the glossopharyngeal, pneumogastric, and spinal accessory nerves. Note that in this illustration the facial nerve is entirely concealed by the auditory nerve. Right figure: Here the facial nerve (vii) has been separated from the auditory nerve (viii) by a blunt hook.

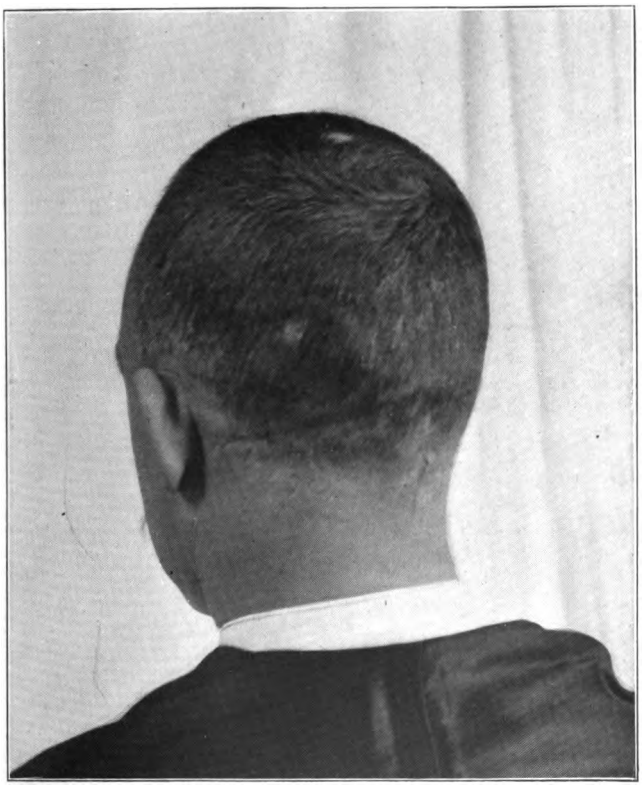

Fig. 2.—Photograph of patient taken two weeks after operation showing scar well concealed within the hair line.

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cies of the problems involved, and yet if one is thoroughly familiar with them and knows how best to deal with them, no apprehension need be entertained as to the immediate outcome.

It is not my purpose to advocate this operation without the fullest realization of its limitations, and yet I feel quite confident that it is based on sound logical reasoning, and has been attended with a measure of relief which fully justifies its performance in properly selected cases.

1724 Spruce Street.

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OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

REPORT OF CASE

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From a clinical and physiologic standpoint, lesions of a limited area, occurring at any level of the brain-stem, are of much interest. Any attempt to analyze the grouping of the afferent impulses in this region has always been attended with considerable difficulty. Impulses arising in the same peripheral system after reaching the first synaptic junction in the spinal cord are formed into groups by specific selection and by gradual filtration away into secondary tracts, and then traverse the cord, grouped according to their sensory qualities, in paths which are frequently isolated by disease.

All of those impulses underlying sensations of posture, passive movement and spatial discrimination, ascend in the ipsolateral dorsal tracts of the cord, grouped in such manner that lesions can not differentiate between the various components. All those impulses of a painful and thermal quality are grouped in the contralateral spinothalamic tracts and can be interrupted separately or *in toto* by very limited lesions. But lesions here make no distinction between pain of a deep or superficial quality. During the passage of these impulses from the cord through the brain-stem to the optic thalamus, the paths of passage are anatomically very closely associated.

Impulses underlying sensations of pain, heat and cold receive the regrouped secondary impulses of a like

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nature from the face and continue their course in paths apart from those conveying the other components, and can be interrupted without a disturbance of other paths, or any one or a combination of any two of these components of sensation may be interrupted in this area without the other one or two being disturbed, or epicritic pain may be grossly involved by the process, while the pressure algometer readings indicate only a partial loss of the deep pain sense over the corresponding areas. In the brain-stem the impulses underlying an appreciation of posture and passive movements take a separate course from that taken by those underlying spatial discrimination, and may be interrupted separately. Among the lesions occurring in the brain-stem, resulting in sensory disassociation is the limited degeneration secondary to occlusion of the posterior, inferior cerebellar artery.

To Drs. Henry Hun[1] and Ira Van Gieson belong the credit of reporting the first case of this condition in America. This case was reported in 1897. Since then Thomas[2] reported two cases in 1907, Spiller[3] two in 1908, Gordinier[4] three in 1911, and Courtney[5] one in 1912. Sixteen cases have been reported in Germany, two in France, and two in England, one by Wilson[6] in 1908, and one by Head[7] in 1911. The few cases reported would indicate that the condition is rare, and I believe that it is so as it is inconceivable that a lesion with such a definite and clearly defined symptom-complex should have been unrecognized, in many cases.

1. Hun, Henry: New York Med. Jour., 1897, lxv, 513, 581 and 613.
2. Thomas: Cases of Thrombosis of the Posterior-Inferior Cerebellar Artery, Maryland Med. Jour., 1907, l, 204.
3. Spiller: Jour. Nerv. and Ment. Dis., 1908, xxxv, 365.
4. Gordinier: Occlusions of the Posterior-Inferior Cerebellar Artery; a Definite Symptom-Complex, Albany Med. Ann., 1911, xxxii, 565.
5. Courtney: Occlusion of the Posterior-Inferior Cerebellar Artery, Boston Med. and Surg. Jour., 1912, clxvi, 329.
6. Wilson, S. A. K.: A Case of Thrombosis of the Left Posterior-Inferior Cerebellar Artery. Proc. Roy. Med. and Chir. Soc., London, 1908-09, ii, 52.
7. Head: Sensory Disturbances from Cerebral Lesions, Brain, London, 1911, xxxiv, 117.

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The following case report indicates quite clearly the character of the symptomatology:

Patient.—J. S., man, aged 45, farmer by occupation, consulted me March 29, 1913, complaining of vertigo, numbness and inability to feel heat over right side, difficulty of using left arm and leg, tendency to rotate and fall to left and difficulty of swallowing. Family history had no bearing on present trouble. Patient had had usual diseases of childhood but no serious illness and no syphilis; drank to excess up to one year previously; had been married twenty years; has five children living, all healthy.

Present Illness.—Patient felt quite well March 13, 1913; ate heartily of pork and kraut at evening meal. He went to sleep early and slept soundly. About 2 a. m. he was awakened with loud roaring sound in head. He felt intensely giddy and vomited; attempted to sit up in bed but was unable to do so; head fell to the left and vertigo was increased. He was fully conscious and observed his symptoms intelligently. His left arm and leg felt paralyzed and the right arm and leg appeared numb. He could not swallow and his voice was husky. He perspired profusely but left face did not perspire nor has it up to the present time. He had horizontal and rotary nystagmus and double vision. His pulse was 42. A physician was called and diagnosed ptomain poisoning. On the following morning patient was unable to stand because of vertigo, could use left arm and left leg with difficulty, had tinnitus and decreased sense of hearing in left ear, had no perception for heat over entire right side below face. He was confined to his bed for about a week but was then able to be up and walk with assistance.

Examination.—I found him to be a well-preserved man of healthy appearance, weight 180 pounds, height 5 feet, 11 inches. Thoracic and abdominal viscera manifested no evidences of disease. The radials were palpable and blood-pressure was 210. Station and gait were both unsteady with tendency to fall and rotate to left, not increased by closing eyes. Motor Functions: Movements of muscles of face good, but expression not so lively on right side of face as on left, left side soft palate paralyzed and uvula pointed to right, swallowed all kinds of food with considerable difficulty. Tongue was projected in median line. Left arm and leg were slightly ataxic but there was no decrease of power.

Eyes: The eyes showed bilateral rotary nystagmus downward and to the left; the left pupil was smaller than the right, both responded equally well to light and accommodation but the left did not dilate so readily nor so thoroughly to shade as did the right. The left palpebral fissure was

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narrower than the right and the left eye was very slightly retracted into the orbit. Fundi of both eyes appeared normal. The tendon and skin reflexes were active but showed no abnormalities. It was in the sensory field I found the chief and most characteristic disturbances. The vision was good and there was no diplopia at this time.

Smell, Taste and Hearing: Sense of smell and taste were not impaired. The sense of hearing was not quite so good in left ear as in the right and he complained of a sensation of buzzing in his left ear.

Spontaneous Sensation: He complained of numbness over the left side of the face, right arm, leg and trunk. His idea of the right arm, hand, leg and foot were not so good as of the left.

Touch: Light touch as tested with cotton wool and Von Frey's hairs was equally good on both sides but patient's appreciation of hair stimulation over the left cornea was not so good as over the right. Pressure touch as tested with the esthesiometer showed no differences between the two sides. He could appreciate the weight of two grams over both sides of face, trunk and extremities.

Localization: There were no differences between the two sides.

Roughness: A sense of roughness was perceived over right face and on both sides over trunk and extremities when the tooth of the Graham-Brown esthesiometer was projected 1 mm., but it was necessary to project it 3 mm. for a perception of roughness over left face. Over right face he could differentiate between a 00 and 0 sandpaper. Over left face it was necessary to use a 00 and 1½ in order that he might perceive a threshold difference.

Vibration: No difference between the two sides.

Compasses: Perception of two points equally good over two sides when applied simultaneously or successively.

Pain: The perception of superficial pain was much decreased over left face and right side below face. Patient had a sense of pain over right face and left side when the prick algesimeter was applied with a pressure of 2 gm., but there was no perception of pain when it was applied to the left face and right side with a pressure of 10 gm. The dragged point of a pin immediately appeared dull when the median line was crossed from right to left face and from left to right trunk. He could differentiate between the head and point of a pin when applied to left face, right trunk and extremities but point felt dull. Pressure-pain sense was considerably impaired over right trunk and extremities. The pressure algometer registered as follows, the readings being in kilograms of pressure: left deltoid, 5; right, 8; left forearm, 9; right, 12; left palm, 10; right, 14; left nipple, 8;

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right, 12; left tibia, 4; right, 8; left sole, 5; right, 10. There were no differences between the two sides of the face.

Temperature: The threshold difference for heat and cold was the same over both sides of the face, but the perception of temperature was not so keen over the left as the right. I was unable to establish a threshold difference over the right trunk and extremities. The patient stated that all temperatures over these areas gave a sensation of cold. His recognition of posture of right arm and leg were slightly diminished and measurements made with the Gordon Holmes apparatus demonstrated that his perception of passive movement was not so good for right arm and leg as for the left. He could perceive flexion and extension of left index with a movement of 2½ degrees but it was necessary to flex the right index 7½ degrees and extend it 9 degrees to evoke a sensation of movement. His appreciation of weight, size, shape, form, texture and consistence did not vary as between the two sides.

ANATOMY

The following anatomic structures are involved in this condition: The posterior-inferior cerebellar arteries are the longest branches of the vertebrals and have their origin from them opposite the lateral surfaces of the medulla oblongata near its middle portion or just about 2 cm. below their union to form the basilar. Each vessel passes outward and backward across the restiform body and between the pneumogastric and hypoglossal nerve roots; it then passes to the under surface of the cerebellum where it divides into two branches, an internal or inferior vermiform branch and an external or hemispheral branch.

The inferior vermiform branch passes backward between the vermiform process and cerebellar hemisphere, supplies the vermiform process and anastomoses with the vessel of the opposite side and the superior vermiform, a branch of the superior cerebellar artery. The hemispheral branch is distributed to the under surface of the cerebellum and anastomoses with the middle and superior cerebellar arteries.

As the inferior cerebellar artery winds around the medulla oblongata it gives off several terminal end arteries which supply the lateral field of the formatio retic-

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ularis and restiform body. The anterior and median areas of the medulla oblongata are supplied by branches of the anterior spinal arteries and the olivary bodies by the lateral branches from the vertebrals. Hence an occlusion confined to either one of the inferior cerebellar arteries would not injure the hypoglossal nuclei or other nuclei on the ventricular floor, the posterior longitudinal bundles, mesial fillet, anterior pyramids or olivary bodies. The cerebellum would be in no wise compromised because of the free anastomosis between this artery and its fellow of the opposite side, together with the superior and middle cerebellar arteries.

The area of softening in the lateral field of the medulla oblongata, as shown by the careful studies of Hun and Van Gieson, Spiller and Thomas, is sharply limited to the formatio reticularis grisea and restiform body and has a vertical extent from about the middle of the hypoglossal nucleus to the point at which the restiform body passes into the cerebellum. Its greatest anteroposterior extent is from the dorsal blade of the lower olive to the restiform body. The parts usually involved are the ventrolateral spinocerebellar tract of Gowers, the dorsolateral spinocerebellar tract of Flechsig, the vestibulospinal and descending trigeminal tracts, brain-stem extension of the spinothalamic tract, cerebello-olivary fibers, nucleus ambiguus, vasomotor and sympathetic fibers and other fibers which pass via the restiform body. The auditory nuclei and nerves are occasionally involved, while the facial and abducents are rarely implicated unless the thrombus extends into the vertebral.

SYMTOMATOLOGY

As stated above the symptomatology of occlusion of the posterior-inferior cerebellar artery is definite and clearly defined. Spiller formulated a symptom-complex from the cases which he studied. A comparison of the symptomatology as presented by the case as reported with his symptom-complex shows some differences. Following are the important symptoms as manifested:

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1. Sudden onset without loss or disturbance of consciousness.

2. No paralysis or paresis of the muscles of the extremities or the muscles innervated by the fifth nerve (owing to the motor paths contained in the ventral pyramids being supplied by the vertebrals and anterior spinal arteries), while the nucleus and axones of the motor fifth are nourished by the basilar.

3. Diminution of the epicritic pain and thermal sense over side of face corresponding to lesion. An algesia with raising of the algometer readings and complete loss of sensation to heat and cold over contralateral half of body below lesion. Paresthesias in the area of disturbed objective sensation.

4. Tactile sensation, intact in all points, diminution of sensation of roughness over ipsolateral side of face.

5. Diminished recognition of posture and passive movement in contralateral arm and leg. All spatial discrimination was perfect.

6. Incoordination in limbs corresponding to side of lesion.

7. Slight cerebellar ataxia with tendency to fall toward side of lesion. This tendency was not increased by closing the eyes.

8. Nystagmus bilateral, more intense when eyes are directed toward side of lesion.

9. Ménière's symptom-complex; vertigo, revolving vertigo, auditory hallucinations, vomiting, deafness on side of lesion.

10. Paralysis of muscles of deglutition soft palate and larynx on side of lesion causing much difficulty of deglutition with loss of phonation.

11. Sympathetic disturbances on side of lesion, causing smallness of pupil, narrowing of the palpebral fissure, retraction of eyeball and loss of sweating in the face on same side. The reflexes were plus but of no special significance.

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SOME NERVOUS SYMPTOMS OF PERNICIOUS ANEMIA

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About seventeen years ago my attention was especially attracted to the distinctiveness of the nervous symptoms occurring in pernicious anemia, and at that time I reported a case before the American Neurological Association in which the pathologic findings were those of a subacute combined degeneration of the spinal cord. At this time I called attention to the fact that the sclerosis was apparently vascular, not systemic in origin. Stengel has stated that this type of anemic patients give the highest blood-count. Bramwell in 1910 reported a case in which the nervous symptoms were characteristically those of disseminated sclerosis and in which the cord symptoms developed three years before the anemia became marked, and he emphasizes the fact that the nervous phenomena may occur a long time before the anemia or cachexia which precedes or accompanies the degenerative changes in the spinal cord. This also has been my observation. Not only is the blood-picture not typical of that in pernicious anemia, but the nervous symptoms usually precede for months or sometimes even years the blood-findings characteristic of this disease. In Bramwell's case before mentioned the anemia was so slight that no blood examination was made and it attracted attention and became a feature of the case only a few weeks before death.

Some years ago I saw a patient in whom the clinical syndrome was that of a chronic myelitis; the nervous

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manifestations overshadowed by more than two years the typical blood-findings of pernicious anemia. Not infrequently the symptoms resemble those of a transverse myelitis or of a multiple peripheral neuritis.

According to Strümpell the nervous symptoms are similar to those of tabes; other writers compare them to ataxic paraplegia. It is an interesting fact that the nervous syndrome may be entirely wanting and yet there may be involvement of the spinal cord. Spiller has shown that the degenerative process may extend into the brain axis.

These symptoms may be purely subjective, such as numbness in the arms and legs. One of my patients complained that the bones were going through the flesh. According to Putnam paresthesias are strikingly noticeable. Knee-jerks may be normal, absent or exaggerated; ankle-clonus and the Babinski reflex may or may not be present; paraplegia may be partial so that the patient may move the legs freely in bed, or it may be complete. Associated usually with this is a loss of control over the bladder and rectum. Lightning pains and objective sensory disturbances are common. Wilson recently reported a case in which gastric crises were present. Dissociation, as in syringomyelia, has been observed. Dimness of vision is quite frequent and occurs early. Optic neuritis, optic atrophy, and hemorrhages associated with retinitis are occasionally seen. Mentality is as a rule unimpaired, although psychotic symptoms have been reported. Camp and others have described cases in which the mental symptoms and signs of paresis were clearly marked.

Dr. Addison, of suprarenal capsule fame, first described (and his description remains classical until to-day) a fatal and apparently causeless anemia which he designated idiopathic anemia, and it was while exploring pathologically the bodies of these patients to find the cause of this disease that he discovered the remarkable lesion which is basic in what we now know

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as Addison's disease. Dr. Pepper taught that this affection is fundamentally a disease of the bone-marrow, the most important red-blood-making structure. On the other hand, Dr. William Hunter contended that pernicious anemia was caused by an excessive destruction of the red blood-cells in the portal circulation, demanding of the bone-marrow an extreme effort, and in these cases, White tells us, the marrow is red like fetal marrow and is evidently in a state of great activity and "that in its hurry to cure the disease it is constantly pouring into the blood immature forms of red cells such as megaloblasts and normoblasts, poikilocytes and red cells staining badly, which give blood films so characteristic of pernicious anemia."

The ultimate cause of pernicious anemia is most likely a toxin absorbed from the gastro-intestinal tract, but as to the exact nature of this toxin we are entirely ignorant. This supposition affords us a plausible reason why in certain cases the nervous symptoms precede by so long a period those of the blood-picture, the time incidence in either case depending on the point of initial attack by the toxin. Marie's suggestion as to the anatomic arrangement of the pial blood-vessels would account for the occurrence of the combined lesion. Its relatively poor blood-supply in health would probably make the dorsolateral cord particularly susceptible to degeneration in abnormal conditions (Taylor). Certainly such an environment is favorable, and as to the selective affinity of toxins, who can say?

The following cases are a fair illustration of the nervous syndrome occurring in pernicious anemia:

CASE 1.—Miss K., aged 55, no nervous heredity, never robust but always well until fourteen years ago, when she had a nervous breakdown, was ill for two months. Two years ago she began to suffer from insomnia and loss of appetite; she complained at this time of a stuffed, bloated feeling in the stomach. She was very nervous and despondent and has continued so until the present time. Vertigo has been constant, worse in the morning; there has been continuous headache,

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the pain extending from the nape of the neck over the head to the forehead. Her eyes feel "as if they were pushed down." She complains of pain in both heels and small of the back, from which it extends to the front of the body and down the anterior aspect to both extremities. There is a burning sensation in the heels as though they were on fire and there is a constant shooting pain in the soles of both feet. Shortly after the legs and feet became affected she began to suffer pain in the hands; it extended up the arms, most severe between the hands and the elbows; it is intense and is of a burning, stinging character. Everything she touches hurts "as if she were touching hot glass." This pain is continuous, never lets up and seems to be steadily increasing. A year and a half ago she first noticed that her walking was unsteady and that when she attempted to wash her face she would almost fall over; she says that her legs feel as if they were wooden and did not belong to her. There is a marked weakness in both lower extremities and she finds it very difficult to arise from her chair without the aid of her hands. There is Rombergism; knee-jerks and Achilles jerks lost; the plantar reflexes plus; superficial reflexes all present with the exception of the gluteal; tactile sense impaired from below the umbilicus. Cold normal, heat exaggerated in the same area; urine normal. Hemoglobin 62 per cent, red blood-cells 4,061,000, white blood-cells, 4,112; color index .8; blood-picture normal, blood-pressure 130 mm. Hg.

Case 2.—Mrs. O., aged 61; father died of pneumonia in his sixty-ninth year, mother of senility in her eighty-sixth year; three brothers living and well, two dead, one of diphtheria, one of acute Bright's disease; three sisters living and well, two dead, one of tuberculosis, one of apoplexy. The patient has had ten children of whom seven are living and well; one died of diphtheria, two died at birth. Climacteric at 48. For the past twenty years she has had a winter diarrhea. She began about five years ago to stumble in the dark. Although the weight is normal, yet there has been a progressive loss of strength; three years ago she noticed that she would tire very easily when walking up stairs; this effort was accompanied by rapid heart action. She complained of "rocking" feeling in the head. She has had three sinking spells lasting about a minute each time, but did not lose consciousness. The legs have grown steadily weaker and there is marked ataxia. She has been confined to bed since November, 1912. Lately she has had difficulty in starting the urine. There is a girdle sensation around the abdomen and a numb feeling in the legs to the hips and in the hands to the elbows. There is partial loss of tactile sense from the umbilicus downward. The loss of sensation in the upper extremities is more marked in the palms of the hands. Patient has a yellow color but says she

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has had this all of her life. Her eyesight has been poor for the past fifteen years.

When examined January, 1913, she was confined to her bed, would lose her legs in bed; there was marked Rombergism, knee-jerks normal, urine normal, hemoglobin 81 per cent., red blood-cells 3,584,000, white blood-cells 3,571, color index 1.1, no nucleated reds but poikilocytosis was marked.

CASE 3.—Mr. A., aged 53; father died of myocarditis in his fifty-seventh year, mother of apoplexy in her sixty-eighth year; a brother killed himself; two sisters are living and well. The patient had diphtheria in his twenty-second year. He denies venereal disease. In 1900 he first noticed that fluid began to gather in the knees and wrists, also in the ankles (hydrops articuli); the accompanying pain was intense. In 1907 he developed arthritis deformans. His face is a lemon color; this has been increasing during the past eight months. Paresthesia in the hands and lower extremities. Impaired sensation (tactile) in both arms from elbows to finger-tips, and in the legs from the lower level of the buttocks down. Aside from Rombergism the neurologic examination was negative. The hemoglobin was 36 per cent., red blood-cells 2,120,000, white blood-cells 5,130, color index 0.9; poikilocytosis, blood-pressure 180 mm. Hg; urine, albumin with granular and hyaline casts. This is the second case I have seen this year in which nephritis was associated with pernicious anemia.

CASE 4.—R., patient of Dr. Hammes, aged 42, mother died of apoplexy in fifty-fifth year, father of senility in his seventy-sixth year; five brothers living and well; one died of chronic nephritis; one sister living, but is asthmatic. The patient had acute articular rheumatism in his fifteenth year. No history of venereal disease. Five children living and well. About three years ago he began to suffer from numbness and a burning sensation all over the body. For the past eight months he has gradually grown weaker, bedridden for three months. In the last three weeks memory has failed notably. At present there is pain associated with cramp in both legs. During the last year the patient has suffered from alternating attacks of diarrhea and constipation. He can move his legs in bed with difficulty. There is marked ataxia, Babinski reflex in both feet and an impairment of all forms of sensation below the umbilicus. Hemoglobin, 46 per cent.; red blood-cells, 2,116,000; white blood-cells, 5,718; color index, 1.1; urine normal.

CASE 5.—Mrs. H., aged 71; father died of cardiac disease, mother of senility in her eightieth year; five brothers living and well; one died of diphtheria; one sister living in frail health, two sisters dead, one of apoplexy, one cause unknown. Two children living and well; two died at birth; there were two miscarriages; one child died of diphtheria. Patient always

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well; climacteric at 48. Suffered from chronic constipation. In May, 1912, she first noticed difficulty in using hands. A cold sensation soon developed in all four extremities, also a tired feeling and loss of power. Patient gradually grew worse and she complained of a distressing numb sensation in heels and knees. There was at times tremor of right arm when trying to feed herself; also aching in the joints, especially of the wrists and in the bones of the forearm. There was a rapid loss in strength and weight. Diarrhea without any apparent cause occurred about every ten days. Profuse night sweats. The pain was so severe and so constant, with marked tenderness over the nerve trunks, that she was supposed to be suffering from multiple neuritis and was sent to us for treatment of that disease. Deep and superficial reflexes normal, urine normal. Hemoglobin, 62 per cent.; red blood-cells, 2,408,000; white cells, 4,280; color index, 1.3; marked poikilocytosis; nucleated reds were present.

Because leukopenia was present in all three cases it is well to recall Türk's statement that in pernicious anemia you must have leukopenia associated with the red blood-cell picture. While there is great reduction in both the red cells and the hemoglobin, this is proportionately much larger in the former, and hence each cell in order to compensate for their paucity has to carry an excess of hemoglobin. A higher color index therefore may be looked on as the constant of pernicious anemia and may be regarded as Nature's attempt at a cure (White).

The direct relation of the nervous syndrome to a diffuse primary non-systemic degeneration, occurring mostly in the spinal cord, has been definitely and clearly established. In my own case, reported before the American Neurological Association in 1896, these changes were most noticeable in the middorsal region; from this point they steadily decreased, ceasing at the third lumbar segment. Commonly the nerve roots, peripheral nerves and the gray matter escape; yet a number of cases have been reported in which the anterior horns were involved. The affection of the blood-vessels is usually slight. This degeneration is, without doubt, due to a variety of toxic causes, namely, toxins from the gastro-

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intestinal tract, poisons, metallic and vegetable (ergot of rye). Without doubt also this pathologic process may occur in persons with a deficient initial life-impulse in whom degenerative tendencies manifest themselves early. Anemia may precede, associate itself with, or follow diffuse degeneration of the spinal cord, but in many instances, notably the cases of Russell, Batten and Collier, there was nothing in the blood to suggest pernicious anemia. In pernicious anemia the anemia, like the cord changes, is a direct result of the toxin, be it a germ toxin or a chemical one, we do not know which. Probably in each instance the personal equation determines whether the pathologic process be a pernicious anemia with or without cord lesion, or a diffuse degeneration of the spinal cord simply. The ultimate facts are that there is a nervous syndrome frequently found in pernicious anemia dependent on a diffuse, non-systemic, primary degeneration of the spinal cord, that this syndrome may antedate by months or even years the characteristic blood-picture, and that both the anemia and spinal cord changes are the resultants of toxic influence. Contrasted with this must be the nervous phenomena incident to subacute, combined degeneration of the spinal cord with which pernicious anemia is not associated.

Byrom Bramwell, in 1874, was struck with the fatty condition of the heart in pernicious anemia, and as he had observed that arsenic was beneficial in cases of fatty heart, he commenced its use in this disease. In 1910 he began the use of salvarsan and has since treated eleven cases with it. Of these four have been apparently cured (remission?), two were strikingly benefited, one case improved at first with ultimate relapse and death, and another patient became somewhat better, but died of pneumonia while under treatment; yet another patient is slightly improved, but still under his care at the time of writing (May 24, 1913), while two patients received no benefit whatever. He regards the intra-

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muscular injection of salvarsan as the best method of administration because it will produce a sustained and continued effect, and he thinks its use in this form much superior to the ordinary arsenical treatment. Neosalvarsan gives much less pain and swelling, and is therefore to be preferred. He uses 0.3 gm. Life being so precarious in grave cases he considers it safer to give the smaller dose. My personal experience in the use of this drug in this disease has been somewhat limited. I prefer the intravenous method and the larger dose, 0.6 gm. In Case 2 the patient was greatly benefited under its use; her nervous symptoms were rapidly disappearing when she left the hospital and she was getting so that she could walk fairly well. Current medical literature gives very conflicting reports regarding the benefit derived from this treatment; much further observation will be necessary before we can properly estimate its true value.

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ABSTRACT OF DISCUSSION

DR. GEORGE A. MOLEEN, Denver: There has been failure on the part of most neurologists, I think, to recognize the importance of blood-changes and their influence on nerve structure. The underlying systemic condition which lowers the resistance of the nerve elements to nervous influences has not been given sufficient attention. We shall, I believe, realize that this is particularly true when we recognize the increased irritability of the nervous reflex centers and the irritability of nerve structures generally in high altitudes, where, if physical factors are considered, we must remember that the amount of hemoglobin, consequently the number of red blood-cells sent to the tissues generally, must necessarily be greater in view of the lessened atmospheric pressure. That nerve structures are vulnerable to influences under anemic states, when otherwise they would not be, has, I think, been generally accepted. For example, it is not uncommon to find that a fatigue neurosis develops more readily in an anemic person than in those persons who might be considered as presenting the antipode of the anemic condition; in other words, in plethoric individuals. About two years ago I saw a number of cases of acute combined degeneration of the spinal cord. These cases have been considered as due to, or peculiarly asso-

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ciated with, blood-changes similar to those of pernicious anemia. I believe, however, the fact that they follow on acute infectious diseases, has been considered of more importance in producing the anemia than that the anemia is the cause of the disease itself. Most of those cases have followed influenzal infections.

Dr. Riggs has emphasized the importance which he attaches to the anemic states as bringing about nervous diseases, and this is a factor which should be given most careful attention. It is an underlying systemic factor in rendering the nervous system of these persons more susceptible to organic change under influences commonly recognized, than that of an individual supplied with a normal hemoglobin content and a full corpuscular count.

Dr. Howell T. Pershing, Denver: I should like to speak of a striking case that comes in one of the groups Dr. Riggs has been discussing. The patient was an able attorney. When about 60 years of age he began to show his age with great rapidity. He became quite pale, his hair whitened rapidly, he lost a good deal of weight and the expression of his face was considerably changed. While working hard at a summer session in Washington, he noticed especially fatigue in the legs. He could walk all right for a short distance, then rapidly became fatigued, but after a rest could get along better. A few months later I had the opportunity of examining him with his family physician. At that time he was resting mentally and having some recreation; he wanted to play golf, but could not walk enough to do that with satisfaction. I could find no sign of organic disease, although he was examined with great care. In the winter he went to Honolulu and there he probably exercised too much. He had a stormy voyage coming back, and on arriving at San Francisco showed some signs of myelitis, having lost control of his legs and showing some sensory loss. He was at that time seen by Dr. Moffett of San Francisco, and later when I saw him he had definite signs of transverse myelitis. There was motor loss and distinct sensory loss, with some disturbance of the sphincters. He returned home and improved somewhat under rest in bed. The point that bothered both his physician and myself, however, was that while we thought this must be a case of pernicious anemia, and treated it accordingly, our examinations of the blood were, as we thought, quite inconclusive. There was only a moderate reduction in the reds, down to something over four million as I recollect, and the number of whites was about normal. The color index was slightly high, about 1.1. There were no normoblasts and there was no poikilocytosis. As time went on there was a continued slow reduction in the red cells, but not enough to account for his symptoms, so far as we could

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determine. Then an obstacle to further study occurred. The patient's daughter had died of pernicious anemia, and he had read much on the subject and studied his daughter's case with the utmost care. He was sensitive and begged to be relieved of the frequent tests of the blood. He had a horror of everything associated with anemia, even of the simple prick of the finger, and we had to respect his wishes. Toward the last week or two of his life the anemic appearance was striking. I think it was one of those cases spoken of by Dr. Riggs, without great changes in the blood. The changes in the cord were clinically distinct. It was the ordinary type of transverse myelitis, coming on with a sharp exacerbation at the time of the Honolulu visit, afterward showing slight changes and a gradual loss.

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CAN RABBITS BE INFECTED WITH SYPHILIS DIRECTLY FROM THE BLOODS OF GENERAL PARETICS?

OBSERVATIONS ON THE RECOGNITION OF THE VIRUS IN THE LATER PERIODS OF THE DISEASE *

WILLIAM W. GRAVES, M.D.
ST. LOUIS

The utilization of the Wassermann reaction and other modern laboratory aids has enabled physicians to recognize more certainly than ever before the syphilitic and syphilogenous diseases and has furnished additional convincing evidence that the heretofore so-called parasyphilitic diseases are manifestations of the syphilitic virus. These laboratory aids have, moreover, thrown serious doubt on the efficacy of our ancient and modern "specific" therapy, on the innocuousness of the syphilitic in every period of his disease and on the reality of the relatively modern conceptions of latency.

Accepting as postulates that syphilis is a chronic infectious disease and that we have no means of definitely knowing its termination during the life of the infected individual, I proposed in a recent communication[1] the theory of relative tolerance as opposed to that of latency. It seemed to me that a period or periods of relative tolerance for the virus, meaning thereby an excellent, a fair, or a poor resistance of the whole or a part of the organism was a better conception, one more in accord with clinical experience, the natural history of

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1. Graves, William W.: Med. Rec., New York, Aug. 24, 1912.

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the disease and the results of treatment than that of latency.

Indeed, the accumulated clinical experience of centuries, the utilization of all modern laboratory aids and refinements in the recognition of the syphilitic, the recurring constitutional symptoms, the recurrence of local and the occurrence of focal and so-called parasyphilitic manifestations support the idea that the virus once invading the tissues of the body as long as it remains never becomes wholly dormant—*latent.*

Certainly, the modern laboratory aids and clinical refinements now utilized in recognition of the syphilitic justify the assumption of many older and modern investigators that somewhere in the tissues of living individuals the *contagium vivum* exists and justify the hope that means may be found by which it may be readily demonstrated in every period of the disease. Nothing short of the actual demonstration of the virus in the various periods of syphilis can satisfy the exacting demands of clinical medicine or lead to the fullest recognition of the protean manifestations of this scourge of the human race. Since the epoch-making discovery of Schaudinn the demand for means of recognition of the virus either in the blood or other tissues or body-fluids in every period of the disease has become more and more insistent, especially so since it was soon found that the blood and lymph-nodes yielded inconstant findings and then only in the early periods of the disease.

Uhlenhuth and Mulzer[2] seem to have been the first to succeed in growing spirochetes in the testicles of rabbits from the blood and blood-serum of syphilitics during the primary and secondary periods of the disease and in one case[3] from the blood of a syphilitic mother four years after infection who had recently given birth to a syphilitic infant. Aumann[4] confirmed their results by using blood and blood-serum from the secondary

2. Uhlenhuth and Mulzer: Berl. klin. Wchnschr., No. 4, 1912.
3. Uhlenhuth and Mulzer: Centralbl. f. Bakteriol., lxiv, 165.
4. Aumann: Med. Klin., 1912, xv, 1710.

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periods of the disease. Nichols and Hough[5] have succeeded recently in growing spirochetes in the testicles of a rabbit from the spinal fluid in a case of relapsing cerebrospinal lues nine months after infection.

Each of these authors emphasized his failure to find spirochetes with the dark field in the material used for inoculations. No one, so far as I know, has as yet recorded successful inoculations of rabbits' testicles or otherwise cultivated the *Spirochaeta pallida* from the blood or spinal fluid of so-called latent syphilitics, paretics and tabetics. Yet work in this direction seems most promising since Noguchi and Moore[6] have recently demonstrated typical spirochetes in a number of brains of paretics. It is the purpose of this communication to give a brief account of an attempt to cultivate and otherwise recognize the virus of syphilis mainly in periods remote from the time of infection and particularly in paretics and tabetics.

My work in this direction began a little more than three years ago with the blood and spinal fluid and now embraces approximately five hundred cases, the majority of which were found in my service at the Alexian Brothers' Hospital. These cases were studied clinically from every angle and Nonne's four reactions were used in practically all of them in arriving at diagnoses. Most of the bloods were collected from forearm veins under sterile precautions while a few were collected in ampules from ear lobules. The spinal fluids were invariably collected in a sterile manner. Bloods and spinal fluids were contrifugalized soon after withdrawal. Blood-serum from corpuscle and serum junction in test tubes or ampules and the centrifugate of spinal fluids were studied in smears stained in various ways, in native preparations, and with so-called vital stains; with India ink and with adequate dark field illumination. In only

5. Nichols and Hough: Demonstration of *Spirochaeta pallida* in the Cerebrospinal Fluid, THE JOURNAL A. M. A., Jan. 11, 1913, p. 108.

6. Noguchi and Moore: Jour. Exper. Med., No. 2, 1913, xvii.

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one case, that of lues spinalis, ten years after infection, were spirochetes found in the spinal fluid with the aid of the dark field or otherwise and in only two cases in the blood and in these during the eruptive stage of the disease.

My observations on the bloods and spinal fluids of syphilitics are wholly in accord with those of many others in showing that the spirochetes can rarely be demonstrated in the blood and spinal fluid. It would seem that until we can acquire more exact knowledge of the morphology of the *Spirochaeta pallida* and are, perhaps, thereby able definitely to recognize the granule forms mentioned by Noguchi,[7] or as it may be an encysted form analogous to that described by Perrin[8] in connection with the *Spirochaeta balbianii,* or the spore stage assumed to exist by many authors, attempts to demonstrate "specific" spirochetes in the blood and spinal fluid with any degree of constancy seem to be hopeless undertakings.

The almost universal negative results in demonstrating spirochetes in blood and spinal fluid derived from syphilitics caused me early in my studies to accept the idea held by many of a cycle of development in which a spore-like stage existed. As early as October, 1910, I found quite constantly in the spinal fluids of syphilitics and seemingly paralleling in number the intensity of the disease processes, non-motile round bodies and actively motile granules. The granules vary in size from ¼ to 1 micron. The smaller granules resemble the smaller so-called hemaconia, have a low refractive index to the dark field, are not centrifugalizable at moderate speeds and are not stained by ordinary dyes. The larger granules have a relatively high refractive index to the dark field, are centrifugalizable at moderate speeds and are readily stained by basic dyes.

7. Noguchi, Hideyo: THE JOURNAL A. M. A., July 12, 1913, p. 1236.
8. Perrin: Arch. f. Protistenkunde, 1906.

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I was at the time and still am unable to find those granules mentioned in the literature as occurring in spinal fluid. They are readily differentiated from fragments of cells found in normal and pathologic spinal fluid. I mention them here merely to caution others against interpreting them as "spores" of the *Spirochaeta pallida*. Continued studies soon showed that similar-appearing granules were found in the blood, in ascitic fluid, in transudates generally, in the bodily secretions such as saliva, tears, bile and urine, not only in syphilitics, but in non-syphilitics and likewise in the blood, spinal and other body fluids and secretions of dogs, rabbits and monkeys.

It finally occurred to me that if the syphilitic virus does remain viable in blood and spinal fluid despite the resistances of the body, then some impetus to its development might be secured by culturing *in vitro* the bloods and spinal fluids of syphilitics. Studies of approximately two hundred bloods and spinal fluids cultured in hermetically sealed ampules failed to yield the *Spirochaeta pallida* in a single instance, though the blood from ear-lobules constantly yielded other micro-organisms, the most frequent being the *Staphylococcus albus*. Moreover, the contaminated bloods as well as those remaining sterile disclosed many forms described by von Niessen[9] as stages in the development of his "syphilis bacillus"; the *"Cytorrhyctes luis"* of Siegel[10]; bodies recently described by W. Ford Robertson[11] as stages in development of his so-called *Bacillus paralyticans* and bodies recently described by McDonagh[12] as forms in the life-cycle of the virus of syphilis. My studies of sterile and contaminated bloods from syphilitics, from non-syphilitics and also from dogs, rabbits, cats and monkeys compel me to say that normal products in the blood, or artefacts, or contamina-

9. Von Niessen: Der Syphilis Bacillus, Wies Baden, 1908.
10. Siegel, J.: Centralbl. f. Bakteriol., 1910, lvii, No. 1, p. 68.
11. Robertson, W. Ford: Lancet, London, Sept. 12, 1912.
12. McDonagh, J. E. R.: Life Cycle of the Organism of Syphilis, Lancet, London, Oct. 12, 1912, p. 1011.

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tion, one or more or all combined, will account for most of the findings in the blood assumed by the authors just mentioned to be either the virus of syphilis or stages in its morphology.

Perhaps the most confusing forms found by me in the bloods of syphilitics and later in non-syphilitics and animals were those which were similar to those described by Krystalowicz and Siedliecki[13] in 1908 as contracted, ring and condensation forms of spirochetes. Indeed, their Forms 12 and 20, and 25 to 47, inclusive, here reproduced in Figure 1. may be readily shown in mammalian blood generally. It can be shown with alkalinized stains, the dark field, with moist India-ink

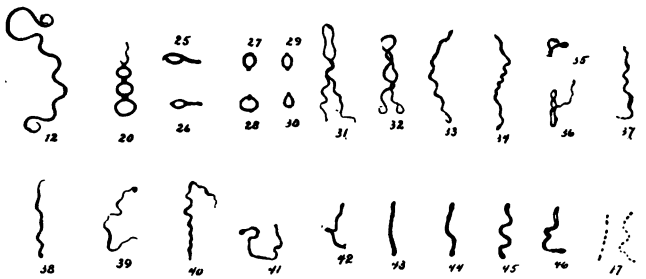

Fig. 1.—Confusing forms found in blood of syphilitics, non-syphilitics and animals; forms that may be shown in mammalian blood generally.

preparations, fresh mounts and vital stains in one way or others that the forms just referred to, as well as several others which I have encountered and at one time considered as phases in the morphology of spirochetes, are either artefacts or normal constituents and these, together with the non-motile round bodies and the granules already mentioned, will be made the subject of an early communication.

With the announcement of Noguchi[14] in August, 1911, of a method of pure cultivation of *Spirochaeta pallida,* two additional avenues of research bearing on

13. Krystalowicz et Siedliecki: Bull. internat. de l'Acad. d. Sc. de Cracovie, 1908, p. 173.
14. Noguchi, H.: Jour. Exper. Med., xiv, No. 2.

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the problem in hand were made possible; first, the morphology of the virus might now be studied in pure cultures free from other biologic products than those in the medium and free from artefacts; second, the medium might prove suitable for the cultivation of the virus directly from blood and spinal fluid of syphilitics.

I have made use of both means and in the beginning largely through the kindness of Dr. Noguchi. In August, 1912, he gave me his C 2 and McD. *pallida* strains and during August and September, 1912, provided me with an abundance of ascitic fluid agar tissue medium, during which months, through the courtesy of Dr. Ewing, I enjoyed the privileges of the pathologic laboratory in the Cornell Medical School. It is a pleasure at this place to gratefully acknowledge my indebtedness to Dr. Hideyo Noguchi for his generosity in supplying me with pure cultures and perfect mediums and to Dr. Ewing for the facilities of a splendidly equipped laboratory.

Both the Noguchi strains have been repeatedly transplanted and studied and no change has been noted in their morphology. The morphology of these strains, as well as of two impure strains which I have succeeded in cultivating in Noguchi mediums, seems to be constant and relatively simple. I have been able to make out with the aid of stains and the dark field but two definite stages in development; namely, the spirochete itself with curves varying from one to thirty or more and spore-like round bodies which were described by Noguchi[14] in his first paper on the pallidum. In referring to his Figures 46, 47 and 48, Noguchi speaks of these spore-like bodies as follows:

> Sometimes several round bodies are seen in a group and these are entangled in a mass of short irregular forms of *pallida*. It is not rare to find a round body connected with one or two young *pallida* as though the latter were just sprouting from the former. The *pallida* with these round bodies are motile. The size of this spore-like round body is variable. They are difficult to stain but when stained they take chromatin stain.

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My observations are in accord with those of Noguchi concerning the form of these spore-like round bodies, their variable size, the sprouts which seem to come from them and their resistance to stains. Only in the best Giemsa preparations may they be seen at all. They are best stained by Stern's silver method. Drawings from a preparation derived from a thirty-eight day old subculture of Noguchi's McD. strain and stained by this

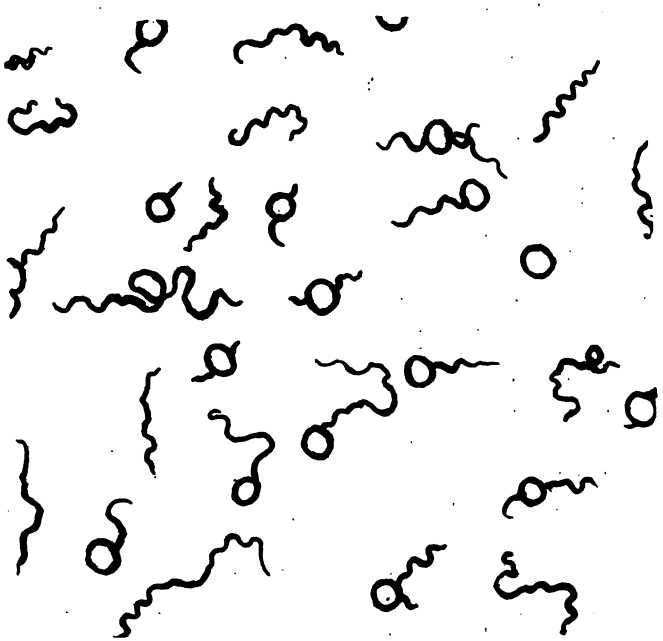

Fig. 2.—Drawings from a silver preparation of Dr. Noguchi's McD. strain illustrating spore-like round bodies and their relations with spirochetes.

method are reproduced in Figure 2. Well-stained Levaditi preparations show them constantly and some in great numbers.

Each of these bodies, as a rule, is spherical, having a uniform rim-like stainable periphery, while its interior seems void and unstainable. The bodies vary in size from ½ to 2 microns. In some cultures they seem to be more numerous than the spirochetes. In others,

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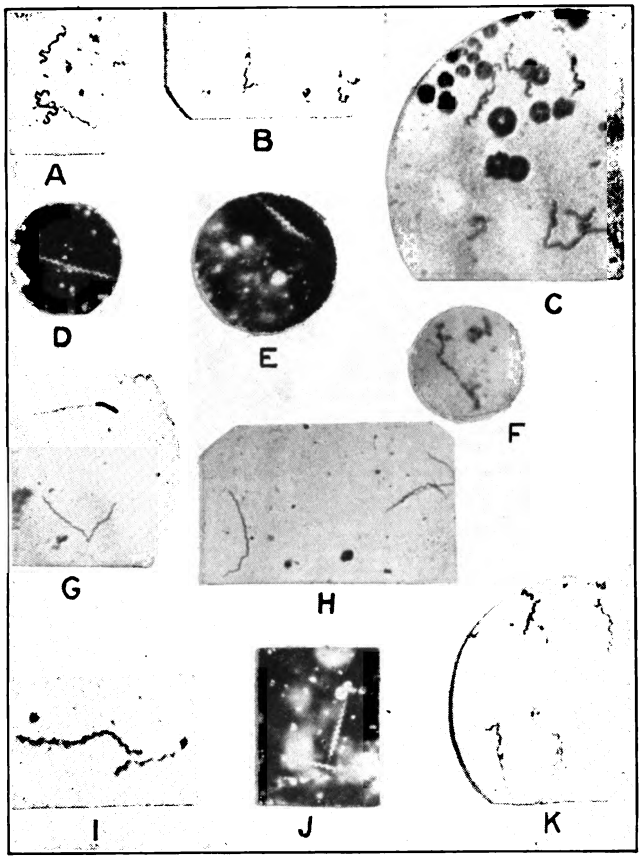

Fig. 3.—*Spirochaetae pallidae: a, b, c, f, g, h, i,* and *k* are from Giemsa preparations from tissue emulsions of lesions of Rabbits 4 and 15. Note the spore-like round bodies in *a, b* and *k*. The others (*d, e* and *j*) are spirochetes from strains 2 B, 2 B X and 2 B X 1 from dark field photomicrographs. Magnification in *i* is approximately 1,000; in other figures 800.

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the converse is true. They are more often connected with spirochetes having few curves. When not sprouting these spore-like bodies are motionless, a point in analogy with spores in general; but in those specimens having one or more sprouts, motility may be made out and this seems to increase with the number of curves. One or more of these bodies are often in juxtaposition, but I have never seen a line of fission.

Noguchi[7] has noted in old cultures the breaking up of spirochetes into granules and I have noted the same in his and in my own cultures. May it not be that these granules subsequently develop into spore-like round bodies? If this be true, then the cycle in development would be: granule—spore-like body; spirochete—granule. Until we learn more about Noguchi's granular forms and the spore-like round bodies and by special stains or other means are enabled definitely to recognize and differentiate them from all other forms, the microscopic recognition of the virus in these and it may be in other forms remains a hopeful problem of the future.

While my attempts to recognize the virus in fresh and cultivated blood from syphilitics in the later periods of the disease have remained negative, I have seemingly been more fortunate in my experiments with Noguchi mediums and with rabbits. With the aid of ascitic fluid agar tissue mediums (Noguchi) I have succeeded in cultivating spirochetes from the blood of two chronic syphilitics, and from the blood of two paretics I have gotten a growth in rabbits.

Experiments with Noguchi mediums were begun in February, 1912, resumed in August, and have been continued up to the present time. The total number of cases from which either blood or spinal fluid or both have been cultured in the ascitic fluid agar tissue medium is 130. Of this number, 40 were paretics; 20, tabetics; 12, lues cerebri; 8, lues cerebrospinalis; 12, lues spinalis; 20, so-called latent syphilitics; 3, pri-

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mary; 8, secondary; 4, tertiary; and 3, congenital syphilitics. The quantity of blood or spinal fluid from each case added to the mediums and incubated at 37 C. (98.6 F.) varied from 0.5 to 2 c.c., and these quantities were passed well down to the tissue with a fine pipet. In a few cases diffuse cultures were made by the addition of syphilitics' blood or spinal fluid in quantities equal to the percentage of ascitic fluid used in Noguchi mediums, and in these cultures no ascitic fluid was employed, the idea underlying this procedure being that greater quantities of the assumed virus might thus be cultured.

A few of the number of bloods and spinal fluids were immediately inoculated into Noguchi mediums, while most of them were incubated from three to ninety days either in hermetically sealed ampules or in test-tubes, and when in the latter, paraffin oil was added to prevent evaporation. It seemed likely that some impetus to development of the assumed virus might be secured by thus incubating the bloods and spinal fluids before adding them to Noguchi mediums. Be this as it may, it is, nevertheless, true that the first culture secured with Noguchi mediums was derived from blood thus incubated. The blood was collected in three ampules (approximately 1 c.c. in each) Nov. 28, 1911, from a normal appearing ear-lobule of a male syphilitic thirty years after "specific" infection. The patient had been admitted to my service at the Alexian Brothers' Hospital, Nov. 26, 1911, complaining of weakness in legs and sphincter weakness. He was greatly emaciated and cachectic, showed definite physical signs of lues spinalis and Nonne's four reactions were present. He died three days after admission, within twelve hours following an intravenous injection of salvarsan. Necropsy was not permitted. The sealed ampules were incubated at 37 C. (98.6 F.) until Feb. 9, 1912, and on this date the contents of one ampule was inoculated into a Noguchi medium tube supplied by Dr. Gradwohl,

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labeled 2 B, and the other ampules were thereafter kept at room temperature.

On Aug. 24, 1912, the contents of a second ampule was inoculated into mediums supplied by Dr. Noguchi and labeled 2 B X, and that of a third ampule was inoculated Nov. 8, 1912, into Noguchi mediums which had been prepared under my supervision, this tube being labeled 2 B X 1. Each of these cultures showed contamination mainly by *Bacillus subtilis,* and each of them many spirochetes, all having the identical morphology, yet differing from the typical morphology of Noguchi's *pallida* strains. The differences thus far noted are (*a*) decided rigidity; (*b*) feeble movement; (*c*) marked regularity in the curves and being slightly wider in the middle; (*d*) extreme resistance to strains.

Cultures 2 B, 2 B X and 2 B X 1 have been repeatedly subcultured without change in morphology and despite every effort at purification the *Bacillus subtilis* remains. The spirochetes in these cultures seem, like Noguchi's *pallida,* to originate from spore-like round bodies, and they have even a greater resistance to stains. The peculiar morphology may be accounted for, possibly, by differences in *pallida* species, or by altered cultural conditions incident to the presence of the bacillus subtilis, or by considering them of a species not hitherto described, or by considering them involuted forms analogous to those noted by Noguchi[15] while cultivating his *macrodentium* strains. Until purification can be accomplished or strains having similar morphology can be secured direct from syphilitic blood drawn under sterile precautions, identification cannot be definitely determined.

The points of special interest in connection with strains 2 B, 2 B X and 2 B X 1 are that the virus was derived from the blood of a syphilitic thirty years after infection and that it remained viable in sealed

15. Noguchi, H.: Jour. Exper. Med., xv, No. 1, 1912.

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ampules for 71, 266 and 310 days, respectively, after drawing the blood and before adding it to Noguchi mediums.

The blood from which the second growth in Noguchi mediums was secured was drawn June 12, 1911, in an ampule from the ear-lobule of a syphilitic twenty-three years after infection. The patient had optic atrophy and diplegia following an attack of lues cerebrospinalis eight years before coming under observation. The blood of this patient was kept at room temperature and inoculated into Noguchi mediums Aug. 29, 1912, one year, two months and twenty-eight days after sealing it in an ampule. This culture was not examined until March 4, 1913, and a few typical spirochetes were found. Several subcultures were immediately made, but thus far no growth has occurred in any of them and the original culture no longer shows spirochetes. In this culture the growth was evidently feeble, but that growth should have occurred at all seems noteworthy, particularly when it is remembered that the blood from which it was derived was kept in a sealed ampule at room temperature for more than a year.

Assuming the strain from the case just cited and strains 2 B, 2 B X and 2 B X 1 to be *pallida,* it would seem that the cultivation of the syphilitic virus directly from the blood of chronic syphilitics with the aid of Noguchi mediums is among the possibilities of the laboratory. Even though the blood from only two cases out of one hundred and thirty yielded positive results, they are, nevertheless, encouraging and should stimulate us to learn, if possible, the conditions most favorable for the successful cultivation of the virus *in vitro* when derived directly from the blood or spinal fluid of syphilitics.

Thus far blood from four classical cases of general paresis and from one case of taboparesis has been injected in quantities of 2 c.c. in each testicle of healthy full-grown rabbits. Only one rabbit has been used in

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each case. The rabbits were free from skin lesions and studies with the dark field showed no spirochetes in the buccal cavities, about the genitals or anus. The bloods were collected in a sterile manner from forearm veins and no visible lesions of the skin were noted in any case. The bloods were neither defibrinated nor was citrate or oxalate of sodium employed to prevent clotting. In four of the five cases bloods were incubated from two to seven days at 37 C.; whereas in one case it was injected two hours after drawing it. Before injecting the blood it was mortared and the clot thus partly broken yielded a rich red serum. In injecting the blood the needle was caused to penetrate the long axis of the testicle, the point of the needle passing twice through the tunics, and as the needle was withdrawn the blood was distributed first between the tunics and, secondly, along the course of the needle. I shall give abstracts of histories and rabbit protocols only in the two cases yielding positive results.

REPORTS OF CASES

CASE 1.—Man, married, aged 37, had sustained a luetic infection eighteen years ago, receiving thereafter two years' continuous treatment. He became a prominent business man and is said to have been in good health until four years ago, when shooting pains developed in the legs, though transient diplopia occurred on several occasions during the last eight years. For the past two years slight unsteadiness in the dark had been noted. Slight change in disposition was noted in the summer of 1911 and in October of the same year a rapid onset of mental symptoms pointing to general paresis developed. He was seen at that time by Drs. Fry, Hoge, Bliss, Herman, Campbell and myself. His mental state was one of disorientation, confusion and expansion.

The physical examination disclosed associated tremor of facial muscles, syllable stumbling, entire absence of lower and upper extremity, tendon and periosteal reflexes, Gordon toe reflex on right, absent abdominals and marked analgesia, especially over legs, slight Rombergism, slight ataxia of lower extremities, well-reacting pupils and normal eye-grounds. The clinical diagnosis at the time was taboparesis. The laboratory findings secured within two weeks after onset were Wassermann reaction in blood-serum and spinal fluid

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positive, 120 lymphocytes per cubic millimeter and increased globulin content in spinal fluid.

Three months after onset a definite remission occurred lasting several months, during which time the patient seemed quite normal mentally. In July, 1912, right-sided epileptiform convulsions occurred, followed by temporary aphasia and weakness. Following this numerous similar attacks with increased deterioration marked the progress of the disease, death occurring in a convulsive seizure in March, 1913. This patient was under active mercurialization from November, 1911, up to within a short time before his death and in July, 1912, he received 0.6 gm. of salvarsan intravenously.

Blood from this patient for Wassermann reaction and rabbit experiment was drawn Jan. 25, 1913. The Wassermann reaction at this time was negative. The blood was incubated at 37 C. (98.6 F.) until February 1 (seven days), when 2 c.c. were injected into each testicle of a full-grown black rabbit, No. 4. No change was noted in the testicles other than transient traumatic reactions until March 6, when a slight induration was found in the body of the right testicle. The induration gradually increased, forming a rather definite nodule the size of a small bean, and on April 8 aspirated material from this nodule showed many typical spirochetes. At the same time loss of hair was noted about the left eyelids, and near the ciliary margins three crusty lesions were noted on the upper and two on the lower lid. On removal of the slightly adherent crusts the lesions were circular in contour, slightly elevated and moist. In addition to these, four similar lesions were found about the perineum and anal margin. The serum from each of the lesions as well as tissue emulsions revealed spirochetes in great numbers. Following the aspiration the nodule rapidly subsided and on April 15 the right testicle was removed. It seemed in every way normal. Neither scrapings from the cut surfaces nor emulsions and Levaditi preparations showed spirochetes. The left testicle which had never shown induration was removed on April 22 and all methods were negative to spirochetes.

The lesions about the lids gradually subsided and by May 2 had entirely disappeared but up to within a few days of this time frequent examinations of tissue emulsions from these lesions showed their presence in great numbers. On May 25 ten discrete lesions were noted on left eyelids again yielding spirochetes. These lesions continued active for about two weeks and again disappeared. The lesions about the anus and perineum persisted somewhat longer and by May 10 had entirely disappeared and they too continued to yield spirochetes until a few days before their complete subsidence. On May 17, one week later, a lesion similar in character to those previously existing developed on the right anal margin. An

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emulsion of this lesion gave many typical spirochetes. On May 18 a superficial erosion was noted about the anal margin yielding in tissue emulsion numerous spirochetes, again disappearing by June 10.

CASE 2.—Man, aged 41, had an initial lesion fourteen years ago and thereafter two years' continuous treatment. He was admitted to Alexian Brothers' Hospital Feb. 3, 1913. Mental change first noted four months ago. On admission marked disorientation for time and place, no insight, expansive delusions, marked excitement, Argyll Robertson pupils, facial tremor, syllable stumbling, unequal and exaggerated tendon reflexes, a pathologic toe reflex on the right. Nonne's four reactions present.

On February 22 blood drawn from forearm vein and two hours later 2 c.c. of this were injected into each testicle of a healthy white rabbit, No. 15. On April 10 (incubation period 48 days) both testicles seemed normal but slightly indurated erosion encircling the prepuce was noted. Both bloody serum and tissue emulsion from this lesion yielded spirochetes in great numbers. This lesion remained active, constantly yielding spirochetes for a period of about four weeks and then gradually subsided, completely disappearing by May 17. On May 27 a discrete lesion recurred on prepuce and continued active, yielding spirochetes for eleven days and then disappeared. No other local lesions have recurred to date (June 15, 1913). Although the right testicle seemed in every way normal, it was removed on April 27. Scrapings, tissue emulsions and Levaditi preparations were negative. On May 2 a slight induration was noted in the body of the left testicle. This gradually increased and three other smaller indurations developed, causing the testicle to become definitely enlarged. This testicle was removed May 22. The indurations were rather white, firm, sharply defined and histologically showed the characteristics of gummas. Tissue emulsions from these areas and adjacent structures failed to disclose spirochetes with the dark field. Levaditi stains are in preparation. On April 10, when the lesion about the prepuce was at its height, Rabbit 15 was placed in a cage with a healthy female rabbit. On April 24 this female, No. F 15 A, showed a definite erosion on left of vaginal orifice yielding serum and tissue emulsions teeming with spirochetes. This lesion continued to show these organisms until May 17 and one week later the lesion had entirely disappeared. The vaginal secretion now as before the mating was found to be free from spirochetes. On May 24 a small erosion was again noted on the left of the vaginal orifice and a bit of tissue again yielded spirochetes. This lesion healed in about ten days and spirochetes have not been subsequently found.

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On April 21, after the vaginal orifice of a healthy female rabbit, F 15 B, was scarified, she was placed in the cage with Rabbit 15. On May 14 two erosions were noted on each side of the vaginal orifice yielding spirochetes in serum and in tissue emulsion. These erosions had disappeared by June 2, but on June 10 the outer surface of the left labium showed extensive erosion again yielding, as had the others, spirochetes in tissue emulsion. This lesion was still active on June 15.

On April 27 the right upper lid of a half-grown rabbit, labelled F 15 C, was scarified and inoculated with tissue emulsion from prepuce lesion from Rabbit 15. On May 24 a moist lesion was noted about the right upper lid yielding positive findings in serum and tissue emulsion. On June 1 a smaller lesion yielding spirochetes appeared on left upper lid near anterior canthus. The lid lesions were still active June 15.

Testicular, corneal, skin and mucous membrane inoculations into healthy rabbits have been made with material from the lesions of each of the infected rabbits. Testicular lesions disclosing spirochetes with dark field have been produced in two rabbits with tissue implantations from Rabbit 4 and in one rabbit with tissue emulsion from Rabbit 15. Attempts to cultivate spirochetes from tissue emulsions from each of the infected rabbits with Noguchi's serum water tissue medium and in ascitic fluid agar tissue medium are being made.

The positive results thus far secured with inoculations of blood from the two cases of general paresis into the testicles of Rabbits 4 and 15 may be thus summarized: After sixty-six days' incubation Rabbit 4 showed spirochetes in right testicle and in tissue emulsions from lesions about eyelids, perineum and anus, the skin lesions persisting approximately forty days. The skin lesions recurred about the anus and eyelids seven and twenty-three days, respectively, after their complete disappearance. . Specific testicular lesions have been produced in two rabbits by inoculating tissue from lesions from Rabbit 4. After approximately forty-eight days' incubation Rabbit 15 showed spirochetes in lesion about the prepuce, persisting approximately forty-five days, and during the activity of this lesion the disease was transmitted to two female rabbits by mating, to the one with, to the other without scarification of

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vaginal orifice; to one rabbit by inoculation of scarified surface of upper lid and to one rabbit by injecting tissue emulsion from prepuce lesion into testicles. A lesion recurred about prepuce in Rabbit 15 seven days after all lesions had completely disappeared. Lesions also recurred in Rabbits F 15 A and F 15 B after their complete disappearance. Gummas were found in right testicle of Rabbit 15 after an incubation period of ninety days. The spirochetes from the lesions in each of the infected rabbits show to dark ground illumination, to Giemsa stain and to India ink a morphology identical with that of *Spirochæta pallida.*

On June 1 tissue emulsion from anal lesion of Rabbit 4 and stained preparations from tissue emulsions from Rabbits 4, 15, F 15 A and F 15 C were sent to Dr. Hideyo Noguchi for study and criticism, and in a letter dated June 7 he states: "I found typical *pallida* both in the emulsion and in stained preparations."

CONCLUSIONS

On the evidence recorded in this paper it seems we are justified in affirming that rabbits can be infected with the virus of syphilis directly from the bloods of general paretics. Until confirmatory and additional work can be done, clinical observations and laboratory methods will continue to support the idea that in paretics, tabetics and so-called latent syphilitics the living virus of syphilis is ever present.

Metropolitan Building.

ABSTRACT OF DISCUSSION

DR. CHARLES R. BALL, St. Paul: Dr. Graves' paper presents positive and definite results which must be seriously reckoned with in any consideration of the nature and cause of general paresis.

DR. SANGER BROWN, Chicago: I suppose it is too early to draw any hard and fast conclusions, but it is certainly very gratifying to know that work tending toward definite demonstration is being done, and I think that it might lead us to hope that we will finally discover the causation of general paresis. There are, of course, many things to be cleared up. A great deal of work must be done by different investigators, and they must compare and check up their results and meth-

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ods closely. For instance, it appears conclusively from Dr. Graves' paper that the specific virus, after remaining in the human subject for twenty-five or thirty years, can infect a rabbit with the positive lesions of syphilis; and yet it is pretty well established, no matter what laboratory experiments might seem to show, that usually after two or three years nothing remains in the human subject which can be transmitted to produce the acute diseases in the human female. These are questions that will have to be cleared up. The main and important element in this work is that it is apparently leading up to the causation of syphilis, and we hope that something may develop from it to enable us to cure or prevent the disease.

DR. WILLIAM W. GRAVES, St. Louis: I hope that I have not made the impression that I feel that my report to-day is in any sense final. The work must, of course, be confirmed by others and must be done on a much larger scale than I have been able thus far to carry out.

I think that the actual demonstration of the virus would convince some people of the real effects of syphilis, not only so far as it affects the individual himself, but also so far as it affects his wife and family. From my own observations I have long since come to the conclusion that the syphilitic, however thoroughly we have cured him, is still a menace to himself and to his family.

In answer to Dr. Brown I may cite this experience, which those who have had opportunity of curing syphilis as I did during the early years of my professional career may verify if they will. Out of twenty cases of syphilis which I had treated no less than two, three, four or five years, as we treated syphilis twenty-five years ago, eight of the wives of the syphilitics gave positive Wassermann reaction, and very few failed to show some physical signs that could be attributed to a syphilitic infection. Out of all of the children of these patients, about seventy, I did not find one that compared favorably with either parent. I therefore believe that the idea suggested by Dr. Brown, that the syphilitic is a harmless member of society after he is cured, is not tenable. If we will observe our patients over a number of years after infection, we shall find that they show ever-increasing signs, particularly in connection with the cardiovascular apparatus and the nervous system, which can be attributed only to a former syphilitic infection. We need to diagnose this condition through a laboratory method. In the diagnosis of the cases we have, of course, the Wassermann reaction, which is of the greatest help, and we need to recognize definitely the virus of syphilis in the various periods of the disease as readily as we now recognize the tubercle bacillus in tuberculosis. I believe that this will be the ultimate outcome of laboratory research in this direction.

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EPILEPSY AND PARESIS IN RAILWAY ENGINEERS AND FIREMEN

CARL D. CAMP, M.D.
ANN ARBOR, MICH.

Although on several previous occasions medical men have called attention to the fact that epileptics and paretics are among those employed in connection with the running of trains and motor-cars and to the obvious dangers incident thereto, the subject has not apparently been given much consideration by the transportation companies, at least, so far as I have been able to ascertain by questioning the medical men they employ.

My attention was attracted to the subject through seeing three cases of epilepsy and two of paresis in the course of two years in men who were employed either as enginemen or firemen on locomotives.

While this is not a large proportion of the total number of cases of epilepsy and paresis seen by me, the points to be made are that these men were engaged in an occupation that made the disease a distinct menace to the safety of the traveling public as well as to their own, and that something could and should be done to prevent their employment in such occupations.

An abstract of the histories of these cases is as follows:

CASE 1.—A. T., a railway fireman, aged 28, came to the hospital Sept. 28, 1912, because of having "bad spells." There was no history of nervous or mental diseases in the family. The patient was single, denied venereal disease, and showed no history of trauma. He did not use alcoholics, but smoked a considerable amount of tobacco.

May 27, 1912, he had the first attack of which he had any knowledge. He lost the power of speech, had a "faint" feeling through his stomach, his arms trembled, and finally

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he lost consciousness. He did not have incontinence, but bit his tongue in the attack and felt weak after it. About three weeks later he had another attack, similar except that he did not bite his tongue. About two weeks later he had still another attack. The attacks were all ushered in by the same aura and none of them occurred on the engine while at work. A neurologic examination was practically negative. The urine, blood and blood-pressure findings were negative and a physical examination was negative. The Wassermann reaction on the blood was positive. An examination of the fundi oculi and of the nose and throat was negative.

During his stay in the hospital he had an attack in which he first became apparently speechless, pointed to one of his companions and mumbled and then fell out of his chair. He bit his tongue and made a few convulsive movements. He was unconscious altogether about five minutes. Following the attacks he could answer questions, but was dazed. He returned to his position because he said that he was about to be promoted to be an engineer and did not wish to lose his place.

CASE 2.—W. B., a railway fireman, aged 27, was admitted to the hospital July 8, 1911, complaining that at times he was irrational and had a headache. His family history was negative so far as could be learned from the patient. He had had only the ordinary diseases of childhood and denied venereal diseases.

The patient stated that the trouble came on in March, 1911, beginning with severe headaches which were accompanied by some visual disturbances. At times he had attacks of epileptic automatism; for instance, he arose at night and ran out of the house, running for almost a mile. At other times he had convulsive seizures. All the attacks occurred in the evening or at night. The neurologic and the physical examinations were negative. The examinations of the blood and urine and the Wassermann reaction were negative.

The case was one of epileptiform attacks, possibly caused by beginning organic brain-disease. He did not intend to return to his work after leaving the hospital.

CASE 3.—V. H. S., a railway fireman, aged 29, came to the hospital complaining of attacks of unconsciousness, April 9, 1911. The family history was negative and there was no history of epilepsy. He denied venereal diseases and used alcohol very moderately.

About nine years before his admission to the hospital he had a fall and, following the fall, a severe headache. About a month after this fall he suddenly became unconscious and remained unconscious for two weeks, after which he apparently recovered completely.

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RAILWAY MEN WITH PARESIS AT STATE HOSPITAL, KALAMAZOO, MICH., FROM JULY, 1910, TO FEBRUARY, 1913

Name	Occupation	Age	Date of Onset	Date of Admission	Remarks
B. C.	Engineer	45	October, 1908	October, 1911	
H. R. S.	Car inspector	37	January, 1907	May, 1912	
S. T.	Fireman	40	February, 1911	January, 1912	Would forget signals and had to be told to shovel coal.
C. F. T.	Engineer	55	June, 1909	July, 1911	
J. L.	Flagman	65	February, 1911	March, 1911	Had allowed a train to run into a street-car 2 weeks before admission.
W. V.	Railroad work	50	November, 1906	December, 1911	
F. M. C.....	Motorman	37	June, 1910	June, 1911	Discharged because of his queer talk.
S. G.	Engineer	31	June, 1911	October, 1911	
W. H. S.	Railroad clerk	52	December, 1905	December, 1910	In 1905 his car ran away from him and he was injured.
E. E. S.	Railroad clerk	53	.....	.....	Nothing of importance.
A. C. H.	Railroad brakeman	37	August, 1911	July, 1912	Nothing of importance in history.
C. L. B.	Engineer	40	November, 1910	October, 1911	Was discharged in October, 1911, after a wreck.
W. H. D.	Engineer	56	March, 1912	November, 1912	

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His trouble began eighteen months before his admission to the hospital with attacks of unconsciousness. In the first attack he was told that they tried to awaken him and could not. It came on while off duty, when he was asleep. The second attack occurred after a hard day's work. He went to sleep and when found was purple, with wide-open, staring eyes. On his admission to the hospital he was having attacks about every two months. They always came on while he was sleeping and he would soil the bed and occasionally bite his tongue. During the daytime he complained of attacks which he described as dizziness. He did not fall in these attacks, but they were actually attacks of momentary unconsciousness, in which he became slightly pale, had a staring expression and would not respond to questions. The neurologic and physical examinations of the patient were entirely negative.

The diagnosis was epilepsy of the grand mal and petit mal types.

CASE 4.—B. K., an engineer, aged 56, came to the hospital June 10, 1911, complaining of nervousness and difficulty in vision with incontinence of urine. His family history was practically negative. The patient denied that he had ever had venereal diseases and said that he had never been ill before nor had he had any injury. He said that he was married and had eight children. He had been working at his occupation up to the time of coming to the hospital.

On examination he appeared very nervous and confused. There was a marked disorientation of time and space. He talked disconnectedly, and could give little or no information regarding his present illness, except that he had had more or less incontinence of urine for two years. He stated that he felt very well; "his only trouble was that his wife wanted him to take black pills and he wanted to take white." He obeyed commands but apparently did not fully comprehend what he was doing. There was a marked tremor of the hands when moving. His speech was typically paretic. His pupils were small, with no reaction to light, and the tendon reflexes of the lower extremities were not obtained.

The diagnosis was paresis, of at least two years' duration before his admission. His commitment to an asylum was advised.

CASE 5.—P. B., railway engineer, aged 46, complained chiefly of numbness on the left side. His family history was negative. He had always been healthy. He did not use tobacco or alcohol and denied having had venereal disease.

His trouble began in February, 1909, when he had an attack in which he found that he could not use his left arm and could not talk. This attack lasted about ten hours, but he was not unconscious. In April, 1909, he had another attack,

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in which he did not lose his voice, however. The attack in this case was over in about fifteen minutes. From then until July he had similar attacks, sometimes as frequently as once a week, was never unconscious and never bit his tongue. In July he had an attack which affected the right side. On his admission to the hospital he had no special complaints but spent nearly all of his time in writing a very elaborate diary and many letters. He had an attack while in the hospital in which he lost his voice, he would not obey commands, and his arms and legs were rigid. He remained in this condition for about half an hour. He had no memory of occurrences during the attack.

The examination showed unequal pupils, with a sluggish reaction to light. The knee-jerks were exaggerated about equally on both sides, but otherwise the examination was negative. The examination of the spinal fluid gave a positive Wassermann reaction and showed an increased number of lymphocytes and an increased amount of albumin. He was overanxious while in the hospital to get back to work on the railroad; he said that the president of the road had written to him and that he wanted him back. He insisted that he felt as though he could work just as well as he ever could.

I am not alone in my experience in this connection. In 1908, Dr. P. C. Knapp[1] called attention to general paralysis as a menace to public safety in transportation, and a committee of the American Neurological Association reported[2], in 1910, on the number of paretics in asylums who had been employed by transportation companies. The committee practically confined its attention to paresis and gave the statistics for a number of hospitals and asylums, mostly in New York and Massachusetts.

Statistics furnished me from the State Hospital at Kalamazoo, Mich., and given in the accompanying table, show that there were eighty-seven patients with paresis admitted from July, 1910, to February, 1913, and of this number thirteen had been employed by railways in positions connected with the operating department; five were engineers.

1. Knapp, P. C.: General Paralysis as a Menace to Public Safety in Transportation, Boston Med. and Surg. Jour., 1908, clviii, 187.
2. Tr. Am. Neurol. Assn., 1910, p. 238.

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Whether or not the occupation has anything to do with the development of the disease I do not know. The constant nervous tension may have some influence in this respect, but the conclusion is certainly justified that there are men at work on railways in responsible operating positions who are epileptics or paretics.

If so much is true we may go on to consider what the result might be. In the report of an accident occurring in the past winter on one of the New York elevated lines, it is said:

> The accident, it has been charged, was due to the fact that the motorman in charge of the second train was not looking ahead, as he should have been doing. This, if true, affords an excellent illustration of the statement made by railroad officials that no matter how excellent may be the equipment of a train, or the mechanical and other devices installed for the purpose of safeguarding the line against accidents, unless the man directly on the job, be he engineer, motorman, conductor, flagman, signalman or brakeman, attends strictly to his duties and has his mind at all times on what he is doing, accidents are likely to happen.[3]

It may be objected that after an epileptic fit no man would be employed further in such a position and that the patient himself would wish to leave an occupation which his infirmity makes dangerous to himself and others. In respect to these points it can be said that frequently these patients do not know that they have epilepsy. They have attacks supposedly due to heat-stroke, indigestion, etc., or the attacks may be of the petit mal type with no consciousness of their existence on the part of the patient and no signs apparent to the casual observer. My own experience has convinced me that even when the patient does know the nature of his trouble, economic necessity may persuade him to take the chance and continue at his work.

In the case of paresis the patient usually has no true insight into his condition. He may complain of some of the symptoms—memory impairment, attacks, etc.—but he usually insists that he is quite capable in his

3. New York Sun, Sunday, March 10, 1913.

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work. The dangers in case an epileptic or a paretic is at the throttle of an engine, or in other situations that require an interpretation of signals and orders and constant vigilance, are too apparent to require extended comment. A certain number of accidents have surely been so caused; whether some of the serious accidents which were apparently caused by a deliberate disregard of signals or orders have been due to the same factor seems not at all unlikely.

There would be but little object in calling attention to this state of affairs if there were no way in which it might be remedied; but this, I feel sure, is not the case. I am reliably informed that all applicants for positions of this character are medically examined before they are accepted, and that for some of these positions repeated examinations of the vision are made. Different railways have somewhat different ways of carrying out these examinations, but no matter how these examinations are made the following points could be provided for:

Since we know that cases of epilepsy and paresis do exist among those to whom the running of trains is entrusted, and since the presence of these patients in an occupation of this kind is dangerous to them, to the railway companies and to the traveling public, it would be to the benefit of all concerned if the medical examinations included careful and frequently repeated inquiries into the presence of symptoms indicating the existence of these affections and, more important still, the use of objective tests, such as those of the pupillary reflexes, best calculated to demonstrate their presence and not requiring too much time in their performance. The selection of the tests to be employed and the value to be placed on each might be determined by an expert appointed by the company, who might then instruct the company's regular examiners either by printed instructions or by lectures, or better still by demonstrations in the use of these tests. A series of such tests might

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be given in a short time, and many of them simply noted in the course of other routine examinations. While such simple tests might not be absolutely diagnostic, they would have some advantages over the present method of diagnosis, that is, waiting for something to happen. If the suspicions of the company's regular examiner were aroused by his inquiries he could refer the man to a consultant who could make more extensive examinations.

924 Baldwin Avenue.

ABSTRACT OF DISCUSSION

DR. C. EUGENE RIGGS, St. Paul: Some years ago I read a paper before the Minnesota State Medical Association on the subject of epilepsy as a menace, and I called attention at that time to the facts set forth by Dr. Camp. I also made reference to paresis, and recommended that, instead of having examination of engineers made simply by a physician or surgeon, there should be a competent neurologist to pass on all these employees whose condition is so vital to the welfare and work of a great railway system. My paper was called forth by a serious accident that happened in the East, evidently the result of a minor epilepsy on the part of the engineer.

DR. C. B. WOODSON, St. Joseph, Mo.: I have seen a number of paretics who were engineers and firemen on railroads Some years ago a man who denied syphilitic history was running a passenger train on one of the most popular roads in our state, when he became violent and was overpowered by his fireman, who stopped the train. This man died a paretic in the state hospital. Another case that impressed me forcibly was that of a conductor on a freight train. He was brought to me by his family physician and I diagnosed the case as paresis, not, however, in an advanced form. The patient finally consented to go to a sanatorium, and he was observed sufficiently long to convince the family that he had paresis. While he was running on the train he had a fight or two with his brakeman, but the latter had superior strength and overpowered him. In reading of various accidents on railroads and of the persons responsible for them, it has occurred to me that possibly the greatest menace is the train-dispatcher. Recently there came under my observation a chief train dispatcher of an important system of railroad in the West, and I found that he had the jacksonian type of epilepsy.

A serious wreck recently occurred near the capital of the state, and the train-dispatcher, who had been regarded as a very competent man, says that he was not well at the time.

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There is absolutely no reason why advanced paresis should not be recognized by any one who hears the patient talk or observes him closely, even excluding the Wassermann, but if the Wassermann is brought to our aid designing physicians and patients can not take issue with it. It would be cheaper and far better if the railway companies would give a little attention to this condition in their men. It would be an easy matter to have them placed in an institution for the purpose of observation.

I believe that if a body like this Section would set forth to the managers of railways the dangers incident to conditions of this kind not only as a menace to life, but also as needlessly subjecting them to suits for damages, they would look into the matter further, and some satisfactory method of examination of employees would be arranged for.

DR. D. S. BOOTH, St. Louis: This to me is an important subject, as I have been connected with railways ever since I have been in practice. I do not think that it is the custom of railway companies to employ knowingly any man who has either of these diseases; but the difficulty is to discover them. Murder and robbery are not permitted by law, but both are still going on. The only method we have of discovering these patients is when they come under observation. When the applicant seeks to enter service the medical examiner is not likely to observe this condition, and the man will not tell him about it. Even after obtaining a history of venereal disease my greatest difficulty is to differentiate the diagnosis of these cases in order to prevent the men from going to work. Coming under the observation of the company's doctors is, I might say, tantamount to discharge. The men know these things and go to other doctors for treatment, and we see nothing of them until something happens perhaps that we have never seen in our service. I do not think that I have even known of an accident occurring as a result of these diseases. Recently a case has come under my observation which several years ago I diagnosed as epilepsy, but from the fact that it was the nocturnal form, and, further, that the patient was supported by outside influence, medical evidence perhaps and probably a brotherhood, and that he was on a switch-engine, he was permitted to retain his position. A few weeks ago he had an attack on his engine. If we do not have the support of the profession we certainly cannot prevent these things, and I, for my part, should be delighted to have any one give me an insight into how to detect these cases early, before the patients show any symptoms or serious accidents occur.

DR. L. HARRISON METTLER, Chicago: I do not think that there is any question but that it is expedient for railway companies to recognize as early as possible a case of disease on account of which life is exposed to danger. A friend

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told me that his father, an engineer on a run of only a few miles, but on a fast train, had figured out the number of things an engineer had to carry on his mind during that time, in order to be ready to meet emergencies. I expected him to say a large number, but was amazed when he told me that there were seven hundred different things to be borne in mind. The results of his investigation were published, I think in *Harper's Magazine*. He remarked that people seldom realize that when an engineer starts out on his run he must at once put his mind on parts of the engine, signals, orders, etc., and that in that short space of time this particular engineer had to carry absolutely in his mind and be prepared to meet about seven hundred emergencies. I raise this point simply to suggest that I have often wondered whether or not the strain has something to do with these conditions, in addition, of course, to actual disease, in some of these cases. Of course, diseased men, we know, ought not to be on an engine. But the strain has a tremendous import in these cases, and there is no question but that a long rest will help even non-diseased engineers.

DR. W. BROWN EWING, Salt Lake: My experience with railway work has been that examinations are perfunctionary. The companies have a long list of questions regarding mumps, measles, vaccination, etc.; the applicant says yes or no; as he perhaps happens to have his legs crossed, one leg is tested; urinalysis is made—and that is about all there is to it. This condition has been largely forced on the companies by the men themselves. Some surgeons of railways are not allowed to strip a patient. They may turn down his shirt in front and examine his lungs with a stethoscope and his heart in the same way. A neurologic examination is never heard of so far as my experience goes. The patients whom I have had on the railways have worked for years without any suspicion of epilepsy, paresis, or any similar disease, until within a short time before they were referred to the neurologist. The labor unions have reached the point now at which they contribute 10 per cent. of the hospital fund and have 90 per cent. of the say as to what shall be done with it. That one feature will, it seems to me, force the companies to do away with collections from the employees entirely, and then treat only cases of injury or sickness in the line of duty.

DR. SANGER BROWN, Chicago: It seems to me that it is going to be a difficult matter for us to suggest anything that would be of particular value in carrying out the purposes which Dr. Camp evidently has in view, that is, the protection of the public and the railway companies. I do not believe that we can do anything of any particular use, as physicians, so far as epilepsy is concerned, because we know very well that there is no extended examination made between the seizures

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to enable us to say with any degree of positiveness that the patient is subject to epilepsy, unless the case has advanced so far that the surgeon can easily detect it. In many cases, however, we can determine with a great deal of certainty whether or not the patient has tabes or paresis.

This matter would not be materially helped by a rigid examination at the time the patient applied for employment. Usually he is a young man then and he would not have the symptoms of either tabes or paresis; after all, the main danger arises from the disease affecting those men who have been in the employ of the company for a number of years and who hold responsible positions. The serious disasters that have occurred have come about in this way: Some trusted employee was attacked with some of the episodes of these two diseases, giving rise to a disaster. We have all examined cases of aged men who occasionally come to us. I recall one case now. A man of about 45, who considered himself in good health, was about to cross a bridge when, as he expressed it, something gave way as though he might have been struck by lightning, shock came over him, he was blind for a few moments and had to be led into a drug-store where he was taken care of. An examination of that case probably a year before would have shown that that man had no knee-jerks and no pupillary reflexes. It is these sudden episodes that cause accident. I recall the case of an engineer who said that the light suddenly went out of one eye while he was running his engine, but fortunately the vision of the other continued until he could get to a place of safety. I cannot see how we could expect to do anything more practical than to recommend some method to protect the public against these episodes, and possibly the public might consider that any action along this line would interfere with public liberty. That is what we hear as soon as we invoke any arbitrary rule. If the employees in responsible positions were required to submit to a thorough examination, say every year or two, it seems to me this would greatly minimize these dangers. I do not think that much would be accomplished by an examination, no matter how rigid, if it were made only when the person applied for employment.

Dr. Leo M. Crafts, Minneapolis: This subject is of great importance. I hope that Dr. Camp's suggestion will be acted on favorably. I think that we should also include the word "taboparesis." I am reminded of a case that I had about twelve years ago, that of an engineer who had been on the road for several years and whose removal I advised because of tabes. He had marked symptoms, could not walk without a cane, had loss of reflexes, etc. I am not sure whether he had diplopia or not while I saw him. In trying to go through the living-room of his house he became lost and had to hunt around to get to the door. He was sent into the country for about a year's rest, and at the expiration of this time he

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came into my office and asked for a certificate to go back on the road. I refused to give it to him, stating that he had no business on the railway again. He was not persuaded, however. I heard nothing from him for three or four years, when he sent his compliments to me, and I found that he was back in the service running a switch-engine in the yards of a western city. He must have been taken back on the railway by virtue of his engineer's certificate, for it does not seem to me that any examiner would pass such a case.

Men occupying important positions in the service should be examined once a year, as Dr. Brown has suggested, and, referring to Dr. Ewing's remarks, we ought to urge the doing away with blank forms of examination. We should make out our own complete, independent report, regardless of any forms. A safety campaign is now being carried on over the country generally—a campaign that applies to the public, but I do not see that it applies much to the officials or others running corporations. If this safety campaign is needed, it should reach the officials of the railways and be taken up in the surgical associations of the different companies.

Dr. Lawrence B. Pilsbury, Lincoln, Neb.: Some time ago there was brought to the Nebraska Hospital for the insane a young man of about 30 who had been employed by a large railway system to operate a switch-tower at a junction in one of the smaller towns of the state. The management of this road is a careful one and few disasters occur, but this man had been working so recently that his last pay-check was sent to him after he had been committed to the hospital. This young man had evidently not attracted attention. The only obvious physical sign he showed was foot-drop caused by injury of the muscles of the foot in childhood, a defect which had never been repaired. The man was apparently an unquestionable paretic, and that he should have been in charge personally of signals and switches, alone in the tower, was obviously preposterous.

Dr. Charles Gorst, Mendota, Wis.: No position connected with a railway is more important to the public than that of the man who runs a locomotive on a passenger train. I believe that a man's personal liberty should not be considered when he is incompetent to fill so important a place. My experience of nine years in the position I now hold, that of superintendent of the hospital for the insane, Mendota, Wis., emphasizes the fact that if proper investigations were made many men who are employed by the railway companies would be found to be incompetent long before the fact is ordinarily discovered. I do not know of any case in which such a man has been found to be incompetent and discharged by the railway company before he has committed some act for which the authorities have taken charge of him and committed him to an institution.

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There is no question but that Dr. Brown's idea of frequent examination of employees by properly qualified men is the proper one. I see no reason why this Section should not do what it can for the protection of the public by passing a resolution favoring such examination. A man who was insane in our institution for years and was transferred as a chronic case to a county asylum was paroled by the superintendent of that institution to his brother and soon after was employed by the railway company and made flagman in the yards through which thousands of people passed on trains daily.

DR. CHARLES W. HITCHCOCK, Detroit: A few years ago I saw a case of well-developed paresis in an engineer who later experienced a remission and so far recovered that he made a trial trip with another engineer; but becoming convinced that he would never be able to gratify his desire, he committed suicide.

DR. GEORGE A. MOLEEN, Denver: I am reminded by Dr. Camp's paper of a case of specific thrombosis. About eight years ago a man presented himself with partial paralysis of one side of the body. In the course of the record it developed that he had been a fireman and was still in the employ of the railway company. He had frequently had lapses of consciousness prior to the onset of the weakness which he experienced, and he admitted that often he lost and resumed consciousness to find that the water level in the gage was considerably below the visible line. It occurred to me then to wonder whether or not some of the explosions that sometimes occur might not be due to lapses of this kind. From this it would seem that it is not only paresis, locomotor ataxia and epilepsy to which we should direct our attention, but also syphilis itself. I quite agree with Dr. Brown that examinations should be made at intervals, but the interval of two years is too long. If I recall correctly, the primary infection in this person was about eighteen months previous to the time of the manifestation of the symptoms. When we recall the lapses of consciousness and the possibilities of automatism and automatic acts on the part of men entrusted with the control of engines and having in their charge the lives of many individuals, we begin to shudder as we contemplate these conditions of mind. I think also we have all come in contact with the conditions in arteriosclerosis in which there are epileptiform seizures with a period of unconsciousness, and these acts are often committed by old and trusted employees.

Would it not also, then, be well to examine the condition of the arteries, as evidenced by blood-pressure, at stated intervals? I think that this is a serious question.

DR. C. D. CAMP, Ann Arbor, Mich.: In considering this entire subject we must also, of course, consider the point of view of the railway company, inasmuch as they do not wish

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to go to any prohibitive expense in making examinations such as the appointment of a competent neurologist might entail. Furthermore, the thing must be done in such a way that the men will not feel that they are continually being spied on.

The man who examines the visual acuity repeatedly could also examine the pupillary reflexes without very much additional time, and he could with an ophthalmoscope take a look at the eye-grounds without consuming much time and also test the knee jerks.

There could also be a simple suggestion to examiners that they should always inquire for dizziness, that they should ask to see the tongue and incidentally look for masses; in fact, those little points which after all have the highest diagnostic value, could be put in a series of instructions to examiners. I think it should be emphasized, too, that we are not making these examinations entirely for the benefit of the public. After all, the employee himself is very likely to suffer in a wreck; and the railway company is certainly going to suffer as the result of a wreck if it is obliged to pay damages. If we emphasize these two factors, the public will come in for protection at the same time.

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PRENEURASTHENIC AND PREINSANE CONDITIONS

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It is with a good deal of hesitation that I again present a subject on which I wrote two years ago. The longer I practice among the nervous and the insane, the stronger is the desire to have a hand in preventing their troubles rather than in curing them. The particular reason for this short paper is to bring out a full discussion of the question of prevention.

For a number of years when examining children I have tried to make up my mind as to what kind of adults they were to become. Results have been most disappointing in the main.

The disappointing thing was that I was not succeeding in finding, among children examined who were evidently not up to an average mental and nerve constitution, any classes or types of temperament to indicate their future neurologic or psychiatric dangers. To put it another way, after I had examined a given child, I found myself quite unable to tell the parents of that child what might be hanging over its head in the way of disease. This has been and is a woful lack. The parents knew and I knew that the child was not average. I am not now speaking of the high-grade idiot or imbecile or even of cases of much-retarded development, but of children who would pass for normal on casual observation, or who were considered as slightly queer. I lacked the knowledge which would allow me to say, "Your

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child has a temperament and constitution which is likely to make him insane or a neurasthenic in the future unless you understand him better and take such and such precautions." The parents had brought the child to me for expert recommendations as to their handling of it. They wanted something accurate and concise. My recommendations were diffuse because my basis for them was hazy. What I needed was a concrete notion of preneurasthenic and preinsane conditions in childhood, just as the tuberculosis expert has his clear idea of the pretuberculous habitus. No such concrete notion could be obtained from the literature at my command, so it had to be constructed. When an adult sufferer from neurasthenia or mental disease presented himself for examination, I went as far back as possible in his childhood history to determine his mental constitution and temperament before his illness asserted itself. I went to both pedagogic and pediatric literature for help.

The result has been the three loosely constructed types which I tried to delineate two years ago,[1] and to which I can add very little at present. My excuse for again presenting them is the importance of the prevention of nervous and mental disease in coming generations. They are not perfect. Perhaps there is no possibility of the general recognition of nervous and mental tendencies the same as we now detect tendencies toward tuberculosis. I want to know the opinion of the Section on this question.

There is at least one other reason for going over this ground again. There has been great activity in the matter of child hygiene among both laymen and the profession during the last few years. Men in general medicine, surgery, and eye, ear, nose and throat work have been studying along with teachers and school authorities

1. Moore, Ross: The Recognition of the Preneurasthenic and the Preinsane Condition in the Young, THE JOURNAL A. M. A., Dec. 23, 1911, p. 2079.

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to the end that abnormalities shall be corrected and future trouble averted. Over the world isolated specialists in neurology and psychiatry are also working. But, as a body, we are doing nothing so far as I know. Is it not time that we should officially record ourselves as trying to prevent as well as cure and relieve disease? To my way of thinking, this prevention can be based only on a better knowledge of child types—the soil on which, in later life, nervous and mental troubles develop. We must get this knowledge first and then we must pass it on to the men in general practice, to teachers, and to parents. The Section on Nervous and Mental Diseases of the American Medical Association is the logical fountain-head for this knowledge.

The three types I have begun to identify are as follows:

The Neurasthenic.—The neurasthenic adult is the logical result of a neurasthenic child or a normal child plus exhausting illness. Natural juvenile neurasthenia is clear-cut. On the physical side its symptoms are a general droopiness, poor standing balance, diminished expression of the face, and fatigue fulness or puffiness under the eyes. When arms are extended one will be held lower than the other and the thumbs will droop away from the rest of the poorly extended hands. Fingers twitch with irregular and slow motion, entirely unlike chorea, when the hands and arms are extended. An effort to stand erect causes an accentuated forward curve of the spine with a relaxed and protruding abdomen.

On the nerve side there is evidence of marked inability to accomplish, which is due to mental and not physical fatigue; an appearance of mental apathy which is not real, but is due to a queer combination of alertness with actual mental fatigue; a full appreciation of events and surroundings with a studied minimum of reactions to them, and abnormal sensibility to external impressions.

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The Preemotional Type.—This type shades into and is often combined with the neurasthenic type. Children of the latter type, however, are usually well formed physically, while the preemotionals may show various physical stigmata frequently found with congenital mental disturbances.

The general distinguishing features between the emotional and the neurasthenic type are to be found in the study of the stimuli to which each type responds most readily. The young neurasthenic is seldom introspective. His stimuli are largely external. The future sufferer from mania, melancholia, hysteria, etc., responds to internal rather than external stimuli. Fear, ambition, love, hate, sorrow—all provide internal reasons for outward reactions, which at times become so prominent as to be labeled "unnatural." His existence is sthenic, not asthenic. He has a large range of interests and he keeps them all going. He is versatile and capable, intense in what he does and thinks, and vigorous in joy and sorrow.

The Predements.—These are the mental shut-ins. They are usually physically well built and admirable, while mentally "un-understood." Apparently happy and care-free, they often carry a hazy and terrifying knowledge of their own apartness from their associates. They are also characterized by a power to carry out their natural aptitudes with ordinary ability or even with brilliancy, thereby covering up defects in reasoning power which may be immense. They are not easily fatigued, not emotional in the usual way, seized by fundamental emotions and held by them because of inability to react to further stimuli; unable to correct ideas by reason and observation, unable either to understand or to explain themselves because of this flaw of intellect which makes impossible the exercise of sound judgment; consciously, uncomprehendingly handicapped.

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These are the three types. As described, the classification is very imperfect and possibly contains serious errors.

Fay Building.

ABSTRACT OF DISCUSSION

DR. C. EUGENE RIGGS, St. Paul: The ultimate fact in this presentation is eugenics. Eugenics, like Banquo's ghost, "will not down." The child is the result of the forces of two ancestral strains. As one author has said, "Heritage is what we are, environment is what we have, training is what we do." Walter has described heritage as a method of reaction of the organism to the environment under which we live. The question is, Can this method of reaction be in any way modified? I believe that it can, Weismann to the contrary notwithstanding. Thompson cites the following instance: As the result of the inbreeding of two wheat plants, one subject to rust, the other immune, the hybrids were all subject to rust; but the inbreeding of these hybrids gave plants subject to rust and plants that were immune in the ratio of three to one. The surprising fact in this experiment, is this: that the plants that were immune to rust ever afterward bred true. This simply emphasizes a statement of Sir Thomas Clouston many years ago that there is an intrinsic tendency on the part of the individual to return to the normal. This tendency is not absolute, however. There is a certain class of defectives on which Nature has put her ban; I refer particularly to the feeble-minded. The nervous system makes its most rapid growth from the fourth to the seventh year. The brain of a child 7 years of age lacks but 2 ounces of the bulk and weight of the adult brain. The brain of a person 18 years of age has the full weight and bulk. Now, from birth until the child is 18 years old we have growth and development, and from 18 years to maturity, 25, we have development. In other words, before 25 we have the nervous system, like Kipling's ship, finding itself—coordinating itself, and bringing the whole organism into complete unity and harmony of action. Then again, from 12 upward we have the evolution of sex; also, somewhere about 14 we have the development of the intellectual, volitional and emotional faculties, the training of which means everything in the life of a child. Further, we know that if there is any weakness in the stock it will manifest itself during the adolescent period. It is at this time that dementia praecox and various forms of manic-depressive insanity develop. Thackeray, Goethe, John Stuart Mill, and many others, passed through a period of depression during adolescence. The important point is that if we can recognize those types which Dr. Moore has called to our attention, if

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we are aware of the dangers, the quicksands, in this period, can we not in some way prevent the cerebral and nervous disaster that otherwise will follow? Certainly we can in a proportion of cases.

DR. G. H. MOODY, San Antonio: We all realize the importance of recognizing early the weaknesses of children in order to anticipate what may develop later. As a general thing, however, I believe that we have a tendency to let the matter drop after our discussions are over, and consequently little has been accomplished. If Dr. Moore can continue and work out a tangible plan to be universally accepted whereby early in the life of the child certain tendencies can be recognized and placed in certain definite groups as he suggests, a great step forward will have been made. If he can keep under observation and follow them up for a few years those cases that he has been observing, he will secure some information that will be of great value and give us something more tangible in the way of classification than we have had heretofore. If by certain groupings which he has suggested we are able to know positively that a certain child belongs to a certain group, and is therefore in danger of developing neurasthenia if not properly guarded, that another will develop the insane complication, and so on, it would help us a great deal in a definite way. I think we are inclined right here to be a little too vague and indefinite in our attempt to do something for these children.

As suggested also, general practitioners should take up the subject, as well as teachers perhaps later. Much has appeared in recent literature regarding the dementia praecox type, which is simply a differentiation of types at a later age. Dr. Moore's proposition to start early in the proper training of these children is a most appropriate and practical one and will prove a valuable addition to the steps taken in handling these cases. If we will closely observe children with these tendencies we shall find that the symptoms manifested even at an early age are in many cases quite definite and readily recognized.

DR. PHILIP ZENNER, Cincinnati: So far as my experience goes I do not know that it would bear out Dr. Moore's ideas but, of course, we do not ordinarily have accurate knowledge of the early childhood of our patients. If further experience should bear out Dr. Moore's ideas, and we could in many cases foretell that this child will develop hysteria, this one melancholia, this one neurasthenia, and this one insanity, there is one great harm that might result from such knowledge. If, as has been suggested, we not only know, but also warn teachers and parents of the dangers that will come to that child, the child might be brought up with the knowledge that this danger is before it; and I can conceive of hardly any-

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thing worse for a child than to be brought up with the knowledge that he is in danger of and must be specially protected from neurasthenia, insanity, or the like. As I say, that is the one practical danger that might ensue from those ideas becoming more clearly outlined in our minds.

DR. G. WILSE ROBINSON, Kansas City: Much can be done by the medical profession, and especially by this particular branch of the medical profession, in the direction pointed out by Dr. Moore. A great deal of good work is being done by school inspection in our cities relative to the physical condition of the children, but practically nothing is being done in the way of examining the mental condition of children in the major portion of our schools. Every city should have a laboratory in which these children could be tested and examined for the purpose of determining their mental condition. We cannot perhaps determine accurately whether the tendency is toward hysteria, melancholia, neurasthenia, or dementia praecox; but we can say that the children are atypical and can advise the parents that there are certain lines along which they can be developed properly—certain lines of study that they can safely pursue and certain lines of occupation that they can follow, while certain other lines of education and occupation they cannot follow. All education is simply the development of attentive control, and when these children develop neuroses and psychoses the cure of these conditions usually resolves itself into education of attentive control. With proper development they will become useful citizens, while improper training makes them public charges.

DR. L. HARRISON METTLER, Chicago: I should like to speak a word of caution on this important subject. The title of Dr. Moore's paper reads "Further Notes on the Preneurasthenic and Preinsane Conditions." I do not suppose any of us will question the desirability of the closest study and careful management of the distinctly pathologic types of these conditions. My suggestion is that, in our pedagogic enthusiasm, we must be careful not to go too far and regard individuals as defective who do not conform to our personal standard of normality. There is a great and serious danger of this among the mild or border-land cases. In the history of the world, the originators of ideas and the instigators of progress have been people who were regarded as being a little "off" in their day. The development of civilization and human advancement are not primarily due to the average, so-called normal, man. The latter is rather conservative and does his part in holding things steady or in preventing retrogression. To the average, normal man, so called, new ideas seem strange, erratic and abnormal. He who brings forth these new ideas is usually atypical as an adult, and may have been atypical as a child; and it is an exceedingly difficult matter at times to differen-

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tiate an individual who is atypical merely in a sociologic sense from one who is atypical in a pathologic sense. Our whole system of public education is evidence of this. Sometimes it were better to let some of these apparently atypical children develop in their own way both for their own good and the good of the world. A dull, uniform mediocrity is not the goal of the human race.

Much here depends on the ability of the examiner. As in meddlesome midwifery, so here there is more danger of too much unintelligent interference than there would be in no interference at all. It may happen that a medical examiner's standard of ability may be quite below that set by the child for itself. The art-loving child may appear to be quite peculiar to the mere card-playing doctor. Again, children are sometimes rendered neurasthenic and hysteroid by their environment and stupid parents. Though the child may here appear to be atypical, it is the environment that is atypical for the child. Someone has well said that if half of the children were dismissed from the public schools and their places taken by their parents, a great improvement would be seen in the child-life of our nation, and, I am sure, much smaller number of atypical children would be met.

Eugenics is teaching us much nowadays. The science is still in its infancy. We must not dogmatize beyond the facts in hand. We must, as individuals and as a profession, learn more what mental health means for a given child and not adopt any fast and set standard, based merely on average mediocrity. This is a large task.

Dr. W. W. Graves, St. Louis: Some one has said that in Nature there is no blind tendency toward perfection, and it seems to me that if we recognize and study types in a comparative way, eventually we shall be able to secure not only definite data by which we may recognize the tendencies of individuals and prevent disasters in their lives, but also information which may be applied in the begetting of offspring in a eugenic way. Let one go into a home for the feeble-minded, into an epileptic colony, or into an asylum for the insane if he will, and there he will meet with essentially different types of humanity from those which he meets in the common walks of life. More than that, if one studies in a comparative way the parents of the feeble-minded, of the epileptic and of the insane, and the near relative, he will often find similar types. The insane, the feeble-minded, the epileptic, are persons whose resistance to disease is inherently lowered; they are unable to adapt themselves to environment, and they succumb relatively early in life. If we can by the study of types learn to recognize what is the underlying factor in disease, if we can determine what are the factors in the life of the individual that cause him to be less resistant

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in the stress and strain of ordinary existence and make him peculiarly susceptible to disease, this will help solve the problem. We must soon recognize the necessity of studying human beings from the biologic point of view. The only conceivable fact underlying every function, whether it be mental, physical, or what not, is that without structure, function is not thinkable. We must therefore study human beings with the same sort of ideals that we study the plants or animals or any other living thing in Nature. While we have no such condition as normal for any living thing, we have ideals of normality, and those ideals are founded on harmony of structure, proportion of structure, and adaptation of structure to function. These factors enter into the point of view from which we should judge human beings. There is an old classical ideal, *Mens sana in corpore sano.* Persons who fulfil this ideal represent the good types of the race, types which we seldom find in insane asylums. Instead we find the abnormal types representing asymmetries, disproportion and disharmonies in development. If we study human beings as we study animals and look for the points of the ideal in their physical make-up, we shall have at once some basis for the determination of the resistances of the individual, not only to neurotic disorders, but also to disease in general.

Dr. Howell T. Pershing, Denver: We have two things to consider here. First, the general scientific facts concerning nervous children, and, second, the tremendously difficult problem of the individual nervous child. It seems to me that most of our work has to be done with the individual, and with him alone. There is a great barrier, as a rule, between the child and his elders. The height and thickness of the stone wall that sometimes separates father and son render it almost insurmountable. The average nervous child is a lonely child. He plays with his fellows, goes to school and talks with his elders, but there is much going on in his mind which he keeps to himself that would be important for his father or the doctor to know. All our conventions tend to repress self-revelation. In being educated into ordinary decency and morals, a child gets a terror of mentioning certain things. He is not going to lay bare his mind before any one, not even his father or mother. It would be a great advantage if to the proper extent we could establish communication between a wise elder—a *wise* one and not any one that happens to be in the schoolroom—and a sensitive, nervous child. With all this, we must respect the personality of the individual. There is a certain amount of reserve that ought not to be broken down. How far shall we go in getting into each individual's own mystery? And in it all we have to be exceedingly careful not to implant any morbid fears in the mind of a child. We must be careful not to give the nervous mother an idea

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that her child is going to be insane or even a neurasthenic, as Dr. Moore has said. This is an extremely difficult matter to deal with. I remember reading long ago a short story by Hawthorne which ended tragically, and, as I recall, the last sentence read something like this: "He had taken the delicate soul of a woman into his rude hands and crushed it." It is easy to take the delicate soul of a child into rude hands and crush it. It is easy to instill into the mind of a child ideas which will act as a veritable poison, and yet there may be ideas already acting as a poison which could be removed if the right person knew of them. So I try to encourage my youthful patients to feel that it is proper and safe to tell me of anything that troubles them in order that I may be able to help them. It is, however, a difficult matter at best.

Dr. C. B. Woodson, St. Joseph, Mo.: Children of the types under discussion should be dealt with not unlike others. To point out that a child is nervous, sending a note to mother or teacher to this effect with suggestions as to care, etc., is simply being meddlesome. The matter should be presented systematically to the parent; it is not necessary to say to the parent that the child will not develop into a strong individual mentally. The important thing for any child is to have good sanitary surroundings, proper nourishment, and a reasonable amount of exercise and play. All children should be taught the importance of bearing disappointment well. If the child goes into a rage because it cannot do the thing it wants to do, it makes no difference whether the condition is one of pre-nervousness or not, it gets the better of that child. Study along lines of special aptitude is perhaps one of the worst things that can be carried out, either in the case of a nervous person or one who is not nervous.

There is no reason why the offspring of a person suffering from a pronounced neurotic taint or history should become insane or be particularly nervous, if the right lines of education are followed out; but the individual who has this tendency or predisposition should not go into a business that would carry with it, for him, a hazard, for this would be sure to cause the development of depression and anxiety. He should be kept on safe ground. When we come to overloading, however, it matters not who it is, I maintain that when the load passes beyond the resisting capacity we can put on enough to crush the individual. Load the horse, the bridge, the wagon, the elevator, beyond resisting capacity, and the structure must be relieved or there will be a catastrophe. The best thing to do in these cases is to establish close relationship of the parent if he is normal. If the parent is not normal it is better not to have this companionship. If the parent is

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so easily startled that the child tends to become timid, this makes the child nervous. If the parent is overexacting this makes the child nervous, although the parent is unconscious of it.

A liberal education regarding matters in general, in school, in business, in dealing with associates, etc., should be maintained as much as possible, and to overcome defects in the child it is highly important for the teacher to cooperate with the doctor to this end.

DR. ROSS MOORE, Los Angeles: The men in general medicine, or those making a special study of tuberculosis, are in a position to say that such and such things really constitute a habitus which predisposes a child to tuberculosis in the future. They can make a clear word-picture of this habitus. Their discussion would not have gone far afield, because there are certain definite ideas in their minds as to what constitutes a pretuberculous condition. A parallel to this is the gist of what it was my desire to bring before you in this paper. Instead of that, we have in the discussion reached a number of different points of view. The discussion indicates the state of mind which is present among neurologists and psychiatrists on the question of preneurasthenic and preinsane conditions. I am inclined to believe that the types which I have tried to delineate are inaccurate. They must be, because they are based on too small evidence. And yet the discussion has been sufficient to make me believe that something along this line might be done if we all went at it in the right way. The subject is a good deal wider than the paper. I wish not only to recognize these things to prevent disease in the future, but also to widen out the question of eugenics to the end that people who are peculiar in these ways will learn not to marry, or, at least, not to have children.

Nature would be a good mother, it is true, if only Nature could have the chance. In these days, however, Nature is so badly modified by what we call civilization, that we neurologists and psychiatrists really have a big field in which to help this good old mother. Interference, of course, which creates panic in the hearts and minds of parents is wrong. If, however—the doctor knows absolutely before he says anything to the parents at all that a child ought to go along such and such a line in his future development—then he has a basis in his own mind for a wise, just, kind, and thoroughly helpful recommendation to the parents.

Most of the adverse discussion which we have had here more or less revolves around the idea of our not doing the right thing by these children and by the parents. The reason

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that we cannot do it is simply that we have not the basis for it. Yesterday we found distributed here a notice from Buffalo inviting all the world to attend a meeting there in August which will deal with the welfare and instruction of children. A few days before I left home communication came to me through the mails giving the names of men all over the world who are particularly interested in this movement toward child hygiene. I was sorry to find few neurologists and psychiatrists among the number.

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